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ARCHIVES OF OPHTHALMOLOGY.

THE PRESENT TREND IN GLAUCOMA OPERATIONS: IRIS-PROLAPSE TECHNIQUE.

BY LT. COL. H. HERBERT, I.M.S., RET'D, BRIGHTON, ENGLAND.

(With two figures in the text.)

THIS is the last of a series of papers[1] on the operative treatment of glaucoma. The thesis already elaborated will be summarized and some practical details discussed.

GENERAL PRINCIPLES.

There is a growing body of feeling that deliberate iris-free fistulization has proved to be a step somewhat in a wrong direction; that it has served its turn in popularizing the so-called filtration operations in general; and that there is a fair prospect of eliminating dangers and defects by spreading out filtration in a line a little away from the conjunctival limbus.

In the last decade it has been conclusively shown that the majority of the primary glaucomas, both simple and congestive, requiring operation in England can be safely relieved for a period of years, and in many cases permanently, by the quite moderate drainage provided by *uniform linear iris-free* filtration. But in dealing with some of the higher grades of plus

[1] *Trans. Ophth. Soc. U. K.*, vol. xxxix. (1919), p. 218, vol. xli. (1921), p. 239, and *Brit. Journ. Ophth.*, vol. iv. (1920), pp. 210 and 550, vol. v. (1921), p. 183, vol. vi. (1922) (February issue).

tension which have lasted for some considerable time, not only would this moderate diffused drainage be insufficient, but the fact must be recognized that it is unobtainable, owing to the tendency to firm healing in these eyes. Apparently in such cases the only way of obtaining anything of the nature of wide linear filtration through the fibrous tunic of the eye is by iris-inclusion.

Hence an extension of the doctrine of different methods for different grades and types of glaucoma and for varying conditions of life. The operations of the future will, I believe, fall into two groups (a) those aiming at linear iris-free filtration, and (b) others acting through wide iris-incarceration or prolapse.

Iris-inclusion operations are advocated for two reasons: (1) with the definite aim of reducing the risk of late infection, and (2) because the relief of tension through impacted iris seems undoubtedly very much more generally permanent than by iris-free filtration. This very important fact seems to have received no recognition hitherto; it may lead to the wide use of iris-incarceration even in mild glaucomas, when the conditions are such that one cannot expect the patients to return promptly for further treatment in case of necessity. The prejudice against iris inclusion is based mainly on old, imperfect, and misleading observations. It is nowadays no more reasonable to class all iris-inclusions together, than it is to group all iris-free filtering scars as equally dangerous. Our aim is to secure safety, not only by widening the area of drainage, but in many cases also by developing protective conjunctival and iritic fibrosis.

In a quite exceptionally large experience of iris-prolapse and incarceration, I have seen sympathetic ophthalmia associated with these conditions only where the uveal tissue had been imperfectly covered by conjunctiva, or where there was almost certainly infection introduced at the time of operation or accident. And ordinary septic late infections associated with prolapsed iris have been fully accounted for by the state of the overlying conjunctiva and the presence of a sclero-corneal opening. That the iris tissue serves in itself directly and materially to aid in the passage of microörganisms inwards must be disputed (*Trans. Ophth. Soc. U. K.*, 1921, p. 247).

PRACTICAL APPLICATION.

(1) *Iris-free drainage.* Some excellent uniformly filtering cicatrices are to be seen after the most varied operations but sclerotomy in some form seems the most rational means of securing the desired result with regularity. Whatever form of incision may be adopted ultimately, my "small flap" operation, aided by very early blinking and finger-pressure in after-treatment, seems likely to make headway during the next few years.

(2) *Iris-inclusion operations.* In many glaucomas during the last few years I have attempted merely to repeat a development which had been observed in accidental wide iris-prolapse following simple cataract extractions and sclerotomies, and which I had copied successfully in a few glaucoma operations at the beginning of the century (*Trans. Ophth. Soc.*, vol. xxiii., 1903, p. 324). A wide subconjunctival fold of iris, bulging evenly and moderately (Fig. 1), gradually loses its color

FIG. 1.

throughout, by de-pigmentation and by the formation of new fibrous tissue, both in the adherent conjunctiva and in the iris tissue. At the same time subconjunctival filtration develops at the sharply bent margin of the partly flattened loop. This allows the bulging gradually to subside later. Aqueous is thus conducted under a fibrous shield to an extended filtering line situated where the conjunctiva is thick and loose. Fig. 2 shows this in diagrammatic form; on p. 13 of

FIG. 2.

the January, 1922, issue of the British *Journal of Ophthalmology* is seen the condition actually present in one of Holth's iridencleisis cases.

Such a result seems ideal for glaucomas judged to be too severe for relief by linear iris-free filtration; but it is not always obtainable. Some eyes are rendered unsuited to this particular method by the effects of former operation. A ring of fine posterior synechiæ may limit greatly the width of the prolapse which can be drawn out, as also may a previous wide iridectomy. And one cannot usually expect fibrosis of a quite small prolapse. Failure to reduce tension fully may be expected if the iris is narrowed and atrophic from glaucomatous changes; the rigid iris-tissue remains impervious to aqueous.

Excluding such cases, however, one may feel reasonably certain of obtaining either fairly general fibrosis or what must be considered the next best thing. That is to say, failing general fibrosis one may expect, by widely diffused drainage under non-adherent conjunctiva, to obtain at least as safe a condition as could be got otherwise. Yet the avoidance of mistakes needs care, particularly in the control of the pupil at the operation (see below); and even apart from mistakes the treatment is at times a troublesome and tedious one. One must keep in touch with some of the cases for months, and small supplementary operations may be needed, either for defective fibrosis or for delayed or imperfect development of filtration.

Where there is not a very good prospect of obtaining general fibrosis, there is little doubt that one should aim at incarceration, as distinguished from prolapse, since the pressure of a small or uneven or perforated prolapse tends to produce the local inequalities in drainage, and the consequent conjunctival changes which we wish particularly to avoid. To keep the impaction flat and low, the one essential is to operate with the pupil well dilated, or to obtain wide dilatation immediately afterwards (see below), and to keep it up for some days. To ensure ample drainage it is perhaps often better to leave the iris uncut, or merely incised; though many excellent results have been obtained by removing a piece of iris and leaving impaction only in each angle of the wound.

Recurrence of plus tension is ordinarily less frequent with wide incarceration than with wide loop-prolapse, probably because in the former case there is less often a complete and intact iris loop.

Speaking generally, it is well unreservedly to accept the observation that unnecessary traumatism of uveal tissue greatly lowers its resistance to infection. Hence the need for gentle treatment of the iris, coupled with conjunctival antisepsis or asepsis approaching that found necessary in cataract extraction.

TECHNIQUE OF THE WIDE IRIS-PROLAPSE OPERATION.

Some of the details discussed below apply to a number of glaucoma operations.

1. *The control of the pupil* at the time of the operation and immediately afterwards is the crux of this particular method. I believe that a perfectly normal pupil would be most suitable —of moderate size, but mobile and resilient. But all of these pupils have been more or less under the influence of eserin. And the time taken to overcome the miosis with 4% cocain instillations, aided generally by adrenalin, varies greatly, depending on the strength of the miotic and on the hardness of the eye. There should be only quite moderate mydriasis, both during and shortly after the operation. In some eyes a few instillations at three or four minutes' intervals may be enough. And in the milder simple glaucomas adrenalin may be instilled once only, at the end, or not at all; while in harder eyes, possibly congested, it may be needed before, between, and after numerous cocain instillations.

Thus the operation can never be a "show operation," sandwiched in among others. The surgeon should operate as soon as the case is ready, as shown by the pupil; he needs to have other work at hand to fill up the time of waiting. If the action of the miotic is not sufficiently overcome, the iris may not stay in the sclero-cornea wound when drawn there. Exceptionally this withdrawal of iris may happen from unexpected rigidity of its tissue. In this case one may have to wait half an hour, or so, for fairly wide dilatation before drawing up the iris again. On the other hand, if the pupil has been allowed to become too much dilated, one may have to wait some time for its partial contraction by 1% eserin instillations. Otherwise the lax edge of the pupil may be drawn through the incision somewhat as in Borthen's operation. Aqueous escaping freely in front of the iris may then produce an irremediably blebby conjunctiva.

I have known leakage of aqueous, sufficient to prevent the desired adhesion of the conjunctiva, to happen from very wide mydriasis occurring very shortly after the operation (from adrenalin and cocain used freely before operation, without atropin), though the pupillary margin was certainly not drawn into the wound. Evidently the contraction and the retraction of the iris towards its base were so strong that the loop of the iris in the sclero-corneal wound remained low and sufficiently taut to prevent the complete filling of the occupied portion of the wound.[1]

In such cases, when it is obvious twenty-four hours or less after operation that fibrosis is unattainable, the use of atropin should be pushed in the attempt to draw prolapsed iris back into the wound and so to produce a flat incarceration.

2. *The sclero-corneal section.* In India during the war I always used a 1*mm* Graefe knife, sliding the conjunctiva and leaving it intact except for the one small puncture. The point of the knife was sometimes used for drawing the loop of iris through the wound, and again for incising the loop subconjunctivally, to obviate the necessity of doing this later. But recently, except where there was some particular reason to fear intraocular hæmorrhage, I have used a broad keratome incision, because the much larger conjunctival opening facilitated the use of iris forceps for drawing up the iris. Where there are no conjunctival adhesions to scleral scars from former operations, I prefer to slide the conjunctiva downwards from a distance of about 6*mm* with the edge of the knife applied horizontally, rather than to dissect a conjunctival flap. A sufficient precaution against accidental limbal puncture is usually the subconjunctival injection of a little normal saline fluid, which should be pressed well down to the limbus. However, a *minute* accidental conjunctival puncture appears to be of little or no consequence, even though it may afterwards

[1] This effect of active mydriasis was well seen in a series of simple cataract extractions which I performed with maximal dilatation of the pupil, obtained by frequent cocain instillations, aided by adrenalin and atropin instillations, the whole spread over ½–¾ hour. The object was to prevent prolapse of iris by this means. This object was not always attained, but the few prolapses which occurred were extraordinarily small; and they were reduced to incarcerations invisible on the surface of the globe, by the continued free use of atropin.

lie immediately over prolapsed iris. After a few days no trace will be seen of it.

A fairly large sclero-corneal section is commonly required, close to the limbus, necessitating the use of the broadest of the three ordinary patterns of bent triangular keratomes.

3. *The prolapse.*—The fold drawn up is usually small, occupying only a half or less of the incision. It can often be enlarged by repeated pressure on the eye, beginning as soon as the anterior chamber has refilled. Light finger pressure through the lower lid frequently produces a marked bulge, with some widening of the prolapse, which subsides again on removal of the finger.

4. The conjunctival incision made by the keratome needs suturing.

5. *After-treatment.*—There is usually no need to bandage the eye. A shield affords sufficient protection, and allows of repeated inspection of the eye during the first few hours by the nurse. Eserin drops, 1%, may be needed more than once to control a tendency to wide dilatation of the pupil. And every opportunity may be taken to apply a little pressure, with the object of widening the prolapse. But even without such attention there is generally a steadily progressive enlargement of the prolapse, unless the pupil be kept very wide.

Twenty-four hours after the operation atropin is usually needed. Fair mydriasis must then be secured; otherwise there may be drawing up of the pupil and the formation of posterior synechiæ. Two or three weeks later there has been a return of plus tension in a few cases. In some cases it has given way rapidly to eserin and massage; but in others the tendency to recurrence has persisted for months, necessitating more than subconjunctival marginal incision with a narrow Graefe knife, to aid in the development of filtration. In the worst of these cases excision of a margin of uveal tissue may possibly be indicated. Should the prolapse obtained be much smaller than was desired, it is better to secure increased drainage by drawing up more iris at a subsequent small operation.

One of these means of obtaining freer filtration may be indicated also to counteract a tendency to the formation of one or more superficial leaking points in an iris-loop which is either too small or unevenly distended.

OPTIC NERVE AND ACCESSORY SINUSES.[1]

By Prof. J. VAN DER HOEVE, Leiden, Holland.

MANKIND is given to inflammations of the nose. Every human being in his turn suffers from catarrh of the nose, one very often, the other less frequent, but nobody rests free of this nuisance, and every time we have it, the inflammation may be spreading to the accessory sinuses of the nose.

When now we bethink ourselves how very close and intimate the vicinity of those cavities to the orbit and optic nerve is, then it seems almost a wonder, that there are still walking around people, who never had any eye disease from nasal origin.

When we take out of the huge mass of eye diseases, which in lapse of time are not without some exaggeration attributed to sinus affections those which concern the optic nerve, then we have to fix our attention on several points.

1. The diagnosis of the optic nerve disease.
2. The diagnosis of the sinus affection.
3. The relations between those diseases.
4. The treatment.

DIAGNOSIS OF THE OPTIC NERVE DISEASE.

This diagnosis is less or more difficult according to the kind of nerve disease. This can be: choked disk, papillitis, atrophy, and retrobulbar neuritis. Choked disk and papillitis, though it is not always easy to differentiate one from the other, are relatively easy to recognise, because the ophthalmoscope reveals already in the very beginning that something is amiss with the optic nerve.

[1] Read by invitation at meeting of the American Academy of Ophthalmology and Oto-Laryngology, Philadelphia, October, 1921.

The diagnosis of the atrophy causes more difficulty at least in the beginning but the most difficulty is caused by the most frequent one, retrobulbar neuritis. The patients sometimes complain of pain behind the eyes in the depth of the orbit especially on movements of the eye. The ophthalmoscope is only of little support to us; in the beginning we see either nothing abnormal or a little hyperæmia of the disk, dark and strongly filled veins, till at last we find the characteristic pallor of the temporal quadrant of the disk. But then it is too late; the affection which could probably be cured before, is then usually irreparable, therefore we have to look out for symptoms which enable us to recognize the affection in the very beginning when the treatment may be more successful.

It is possible that in lapse of time the examination of the light sense will help us, till now this is not yet studied enough to be of much value and the best indices are given by the examination of the visual field. Here we find sometimes as atypical symptoms, concentric contraction of the visual field or annular scotoma and as characteristic signs: central scotoma and enlargement of the blind spot.

Till now we do not know why the first loss of function appears in the peripapillar and central fibers; we have only to accept the fact.

We know that both scotomata begin as relative, *i.e.*, for colors only, whereas later even the white light is not observed. Both scotomata can, when they enlarge, flow together and cause in this way the well-known oval scotoma including and surrounding the fixation point as well as the blind spot.

As a rule enlargement of the blind spot appears first: if only one of the scotomata develops, it can enlarge till it invades the region of the other and causes the same oval scotoma, including blind spot and fixation point.

If we find this scotoma we cannot say in which way it was formed, but only that the peripapillar and the central fibers are affected.

Of course when we find one of these symptoms we must exclude the possibility that it has any other origin; the central scotoma can be caused by degeneration or hæmorrhage in the macula, etc. The peripapillar scotoma occurs in high myopia, medullated fibers, hysteria, etc.

Both symptoms are very important and we know with certainty that they can be caused by sinus affection. There are cases of enlargement of the blind spot, as well as of central scotoma, where the scotoma decreases or vanishes by treatment and comes back with every relapse of the sinus disease.

We find optic nerve affection more frequent in diseases of the posterior than of the anterior sinuses; in the latter they are rare.

These symptoms are important as they are not symptoms of a sinus affection but of an affection of the optic nerve. They show that the optic nerve is diseased but do not teach us anything about the origin of this affection and we always must remember that every retrobulbar neuritis can begin with these symptoms whether it is caused by multiple sclerosis, by tuberculosis, syphilis, dental infection or rheumatism, or by nasal disease.

So we come to our first conclusion:

THE OPHTHALMOLOGIST HAS IN THE EYE NO SIGN AT ALL TO DISTINGUISH THE ORIGIN OF A RETROBULBAR NEURITIS.

DIAGNOSIS OF THE SINUS DISEASE.

Every rhinologist will be convinced that the diagnosis of an existing sinus disease especially of a posterior sinus disease is not always easy, nay, we may say, not always possible.

So we find in Mackenzie's *Diseases of the Throat, Nose and Ear*, edited in 1920, "The absence of any sign of disease inside the nose is not sufficient to exclude sinusitis from the diagnosis."

With the purely rhinological methods of examination we are not able to diagnose a great number of sinus affections; happily we have a good support in the examination with Röntgen rays. We can photograph the sinus in different directions: occipito-frontal, when we wish to compare both frontal sinus, bitemporal and occipito-caudal according to Pfeiffer's method to compare both sphenoidal sinus.

For the ethmoidal sinus I always think the best way is the method of Rhese. We let the patient lie down on his tuber frontale, nose and ansa jugularis, and put the tube above the protuberantia occipitalia. Then we get in a normal person a

photograph where you see the frontal sinus, the orbit, and the region of the ethmoid; by this method we ought always to compare both sides.

For the ophthalmologist it is of great interest that we can get in this way an excellent image of the optic foramen and the fissura orbitalis superior, so that we can find out if something is amiss with the optic foramen. You see on these different photographs that the foramen is on the Röntgen photo a definite round hole.

We have photographed in this way many persons with fractures of the skull bones and with optic atrophy to see if we could find the reason of the latter; so you see here the callus on the base line of the skull and an optic foramen which has an hiatus at the upper side and a fissure running from the optic foramen in the skull. Here a broken baseline and a callus at the optic foramen; the baseline can also be broken by other processes as for instance by a gumma as in this case from Sonnenkalb. Here you see an optic foramen which has quite another shape after a skull trauma. I use this method also to see whether in patients with tower skull the optic foramen is free. It is said that the optic nerve affection in tower skull is possibly caused by a deformation of the optic canal and Schloesser even operates on this theory to free the optic nerve in the canal. If now I find a normal foramen I do not believe the canal much deformed and I found a normal optic foramen even in cases in which there was a mighty tower skull with choked disks.

Rhese's method shows us affections of the frontal sinus as you see in this case of an osteoma which overshadows the optic foramen, but the great advantage of the method lies in the examination of the ethmoid. So you see here obscuration of the ethmoid as in posterior ethmoiditis. How important this method can be is shown by the following case:

A physician had retrobulbar neuritis and choroiditis; he was operated upon for sinus disease several times by one of the best known rhinologists and who told him, that though he did believe that he had not opened all affected ethmoidal cells he did not dare to go further.

This man visited me in Gröningen, I found a huge enlargement of the blind spot especially for colors and a hyperæmic

disk. I advised him to have his nose treated again. The rhinologist, however, thought him to be a neurasthenic and did not wish to operate any more. The man went to Dr. De Kleyn in Utrecht. Dr. Stenvers made the Röntgen photo I show you here, and you see the whole ethmoidal region is clear except a rectangular spot close to the optic foramen and superior orbital fissure. De Kleyn opened at that spot some posterior ethmoid cells in which he found purulent secretion and from that time the man was relieved of every complaint.

So we see the Röntgen photography may be of great aid in the diagnosis of sinus disease and yet there are cases which are not found out even with Röntgen photos. The best proof of this is that from the nine known cases of mucocele of the sphenoidal sinus in no case was the diagnosis made before the opening of the sinus.

In my case three rhinologists, among them the professor of rhinology in Leiden, declared that they could not find a single sign of sinus disease. The Röntgen photograph was, as you see, not clear. Gerber supposed that this is caused by the extreme thinness of the bones surrounding the dilated sinus. When the professor of rhinology opened, on my advice, the posterior cavities a yellow green mucous fluid streamed out and now we could with a probe not only reach the roof of the sphenoidal sinus but find out that the roof was already perforated and that the sinus was in communication with the cerebral cavity.

Mackenzie is also of the opinion that even the Röntgen ray examination is not always sufficient, for he writes:

All cases must be submitted to an X-ray examination by a radiologist experienced in the skiagraphy of the nasal sinuses. But even X-ray examination is not infallible, so that in the presence of serious orbito-ocular trouble, such as optic neuritis, for example, the only satisfactory method of excluding suppuration of the sphenoidal and posterior ethmoidal cells, the sinuses most likely to set up eye disturbances, is to operate and open them up.

So we must say as second conclusion:

THE RHINOLOGIST CANNOT SAY WITH ABSOLUTE CERTAINTY THAT A PERSON HAS NO SINUS AFFECTION.

RELATIONS BETWEEN OPTIC NERVE AND SINUS DISEASE.

How is it possible that the optic nerve is affected in sinus disease?

To get an idea of this, we require an histological examination of sinus, optic nerve, and surrounding tissue in such cases, and it is a great pity we possess only very few of these.

As a rule patients do not die from sinus disease, and when a patient with sinus and optic nerve disease dies, an autopsy of the skull is rarely obtained.

We have but little chance that the scarce quantity of histological material will be increased.

This chance will be better for material from sinus tumors than from sinusitis, though the latter is more frequent.

Happily we have at least three histological examinations which are all very important: two of tumors of the sphenoid and one of a pansinusitis.

It is highly interesting to compare the first two; in both cases there could be observed in vivo an axial neuritis which caused a central scotoma for several months.

Birch Hirschfeld found in his case, typical degeneration of the maculo-papillar fibers present, whereas on the contrary De Kleyn and Gerlach found in their case, though the central scotoma had existed for seven months, nearly no change in the optic nerve, they only observed the veins and capillaries of the optic nerve and nerve sheaths to be more filled than usual and a little infiltration of some nerve sheaths to be present.

These two cases are typical for the clinical processes of the optic nerve disease in sinus affection, for we know we must distinguish here two forms, one which improves directly after opening the sinus, the other in which improvement does not take place or only partially.

In the former there can be, as in the case of De Kleyn and Gerlach, only small and reparable changes in the optic nerve, in the latter irreparable changes are present as in Birch Hirschfeld's case.

The clinical symptoms in the reparable cases may be caused by œdema, toxines, pressure and beginning inflammation; in the irreparable by degeneration and atrophy as a result of too long existing inflammation or pressure.

De Kleyn and Gerlach's case is important, because it shows that even after a seven months' central scotoma nothing irreparable was found so that we must never despair, though the duration be ever so long, but treat the case as soon as we get a chance.

The other case of De Kleyn and Gerlach shows us how an inflammation can extend from the nasal cavities to the optic nerve and penetrate the bony separating wall. They found on histological examination in a patient with pansinusitis with ulcerations in the nose and tear passages, infiltration around the epithelium of the mucosa of the sinus and they saw, as I show you here, the infiltration penetrate in the marrow holes of the bone and in other spots the infiltration follow a little vessel. By an unlucky accident some of the specimens were lost just from the region where the infiltration reached the optic nerve sheaths; the other cuts are so clear and distinct, that we see how the inflammation extends from the sinus to the optic nerve.

The optic nerve sheaths are infiltrated just at the spot where they are nearest to the sinus and we see that an interstitial optic neuritis starts from the nerve sheaths.

The anatomical disposition may be of great influence whether an optic nerve will be diseased in the course of a sinus affection and we know, thanks to Onodi, that the anatomical relations may be very different.

Onodi showed us that the optic nerve can pass unprotected by any bony shield through an ethmoidal cell or even through the sphenoidal sinus, whereas on the other hand in some cases the optic nerve is surrounded by a thick wall. This of course can be of great influence. If, for instance, a cell or sinus is filled with pathological secretion and the exposed optic nerve is surrounded by this fluid, the nerve can be affected; if there is only a little or no secretion at all, the nerve can become diseased by direct contact, because it passes twice through the affected mucosa.

On the other hand a swelling of the nerve from toxic œdema may cause more disastrous effects if the nerve is enclosed in a bony canal, where the swelling may even give rise to such a pressure that choked disk or atrophy is the consequence.

The bony separating wall is not an absolute protection

against invasion and in the case of De Kleyn and Gerlach, where pansinusitis gave rise to an optic nerve disease, we saw that the inflammation can penetrate the wall and cause an inflammation of the optic nerve sheaths and of the nerve itself. We cannot say, that the protection of the nerve by a bony wall is always an advantage.

As a rule the chance for the nerve being affected depends on the vicinity of the diseased sinus, and a sinus which touches the optic nerve of both sides, or of the opposite side only, can give rise to affection of both nerves or of the opposite nerve. The many variations that are possible in the relation of optic nerve and sphenoid sinus are shown by the drawing of Quix and the preparations of Onodi.

The following ways, in which a diseased sinus can affect the optic nerve, are possible:

I. By direct spreading of the inflammation as is shown by De Kleyn and Gerlach's case.

II. By pressure by the walls of a dilated sinus as we see in the cases of mucocele where every nerve in the neighborhood may become atrophic by pressure.

III. Deleterious influence may be exercised by toxines, œdema, congestion, etc.

We distinguish:

(a) *Reparable* optic nerve affections, which can exist for months and months, caused by toxines, œdema, congestion, slight inflammation, slight pressure, and

(b) *Irreparable* optic nerve changes caused by degeneration and atrophy, following the same processes as mentioned above, but in stronger degree.

For a better understanding of the way in which a sinus disease involves the optic nerve we stand in great need, not only of more histological material of sinus and optic nerve from autopsies, but also of bacteriological and histological examinations of the material, which the rhinologist obtains at operation.

THE TREATMENT.

Here we have to distinguish those cases of optic nerve affections, in which we find a sinus affection and those which

are of unknown origin, where no sinus disease can be diagnosed. The first cases are easy enough. When we find in a patient with an optic nerve disease a sinus affection present, we have to treat it. We know that a sinus disease can cause an optic nerve affection and therefore we have the right to suppose that if a sinus disease is present in the same patient with an optic nerve affection of another origin, the latter can be aggravated by the former. Where this is the case we ought to treat every sinus affection in a patient with optic nerve disease even if this is not of nasal origin. I always advise ophthalmologists and rhinologists to begin their treatment in a conservative way. Many sinus diseases are cured with a cocaine-adrenaline spray. The other group causes us on the other hand very much difficulty.

What is to be done when a patient has an optic nerve disease and the rhinologist does not find by examination, Röntgen rays included, the signs of sinus disease.

Of course the patient must be examined carefully for internal and neurological diseases. When some cause for optic nerve disease is found, I should still always warn: mind the accessory sinus. It is possible that a patient with multiple sclerosis, with diabetes, syphilis, tuberculosis, rheumatism, or post-trauma has a sinus disease and it could be very disagreeable were we contenting ourselves with one cause for the optic nerve affection and overlooking the sinus disease which might be the real cause or an adjuvant. I remember a patient who was sent to me by an ophthalmologist, because he had optic nerve disease and purulent secretion in the nose, but the rhinologist refused to open the posterior sinus, because he did not find signs of sinusitis and the patient had syphilis, so that according to the rhinologist the origin of the nerve disease was absolutely explained by the syphilis. So they waited till one eye of the unlucky patient, who only got antiluetic treatment, was absolutely blind, the other almost. In Leiden the rhinologist could not find any purulent secretion in the nose but when on my advice he opened the posterior sinus of both sides there was a purulent inflammation of both sphenoidal sinuses and of the posterior ethmoid. Soon after this the visual acuity of one eye recovered to better than ½ but several scotomata remained and the other eye stayed blind. Not only can a

patient with multiple sclerosis, syphilis, tuberculosis, rheumatism or trauma suffer from an independent sinus disease, but he can have a sinus disease caused by syphilis, tuberculosis, rheumatism, or trauma; so that these diseases are not the direct cause of the optic nerve disease but the indirect, whereas the sinus affection is the direct cause. We ought always to examine the nose and accessory sinuses in every patient with optic nerve affection even if there is found another possible cause for this affection. When nothing at all is found, then many authors say the case is one of multiple sclerosis because this disease is in 50–70% the cause of a retrobulbar neuritis and the optic nerve affection may precede every other symptom 10 and more years. Even if this is right we cannot let our treatment be influenced by this consideration. When there is an optic nerve disease without any other symptom of multiple sclerosis, then for us the patient has no multiple sclerosis, at least we cannot perceive it and we have to treat him till he gets other symptoms of this disease. The question what to do in these cases of optic nerve disease without known origin is very difficult and they necessitate a very close coöperation of the rhinologist and the ophthalmologist. We see in other countries how necessary people think it to discuss this problem in joint assemblies of rhinologists and ophthalmologists; they did this in September of last year in Germany in Nauheim; in this year in April in Vienna, and in May in Paris. These joint assemblies were found so useful that the Société Française d'Opthalmologie determined to have such a joint assembly every year. You Americans have the good luck to possess this Academy so that you can discuss this and other problems together as often as you like.

Now there are ophthalmologists who say that when the ophthalmologist thinks it necessary that the sinuses be operated upon the rhinologist has to obey and operate; he is in this way the handcraftsman for the ophthalmologist.

I am not of that advice! I think that an organ specialist is not only the man who knows most of his special organ, has the most practice in operating on it, but is also the man who has to guard over his organ as a precious treasure, which is under his trust, and prevent every unnecessary mutilation.

No surgeon has the right to operate, who means to mutilate

an organ, unless thoroughly convinced, that by what he is doing he is giving the best chance to the patient; he never can do any operation on the responsibility of another doctor. The case is the same in appendicitis, ulcus ventriculi, trepanation for tumor cerebri as in sinus disease. Of course the surgeon cannot master every special method of examination and so he has to rely on the internist when he tells the surgeon what he found by the examination of the stomach and intestinal contents, and the ophthalmologist when he tells him of the visual acuity, the visual field, the choked disk, the neuritis, etc., but then he has to examine the patient himself. When both have communicated what they found by their examination and what they think of the diagnosis then they should consult about the possibility and the desirability of operation in general and in every individual case. If the surgeon is then convinced by his own examination or by the arguments of the other doctor, he has the right to operate, otherwise never.

To return from those general remarks to the optic nerve and sinus disease our treatment will be greatly influenced by the above mentioned considerations.

1. The ophthalmologist cannot say whether an optic nerve disease is of nasal origin or not.

2. The rhinologist cannot exclude with certainty the presence of a sinus disease.

3. Opening of the sinus even if nothing pathological is found in the sinus may be of passing or permanent benefit on the optic nerve disease.

I think this improvement results from the bleeding or a change of the lymph current, in other words it acts like bloodletting or as the instillation of dionin or as subconjunctival injections.

When we ask the ophthalmologist if the sinus should be opened and he views the question only from his narrow point of view as organ specialist, he must say yes as the eye has nothing to fear from such an operation and much to gain, whether a sinus disease is discovered or not.

If on the other hand the rhinologist is asked: must we operate? he must say as organ specialist no because the nose is mutilated by the operation and there is only little chance that the nose will gain anything as is the case when a sinus

affection is discovered. The two specialists then should consult together, not in the way described by Eicken, where the ophthalmologist says: this patient will soon be blind if you do not open the sinus and his eyes will be saved if you do. Then the rhinologist has no choice but he is persuaded by unfair means for no ophthalmologist can say whether a patient will get or will stay blind, and that he will recover after operation.

Our treatment will of course be influenced by the eye symptoms; when a patient is blind or nearly blind no rhinologist will refuse to give him a last chance, but the greatest difficulty lies in the chronic cases.

Here the rhinologist must ask the ophthalmologist what can be gained by operation? What does the ophthalmologist know from literature and from personal experience about the frequency and the degree of recovery of sight after operation and in what number of cases is a sinus disease found present? On the other hand the ophthalmologist has to ask the rhinologist what damage is done by operation aside from the possibility of an optic nerve exposed in a sinus being injured by an imprudent operation. In what percentage does damage occur after a sinus operation and what is its nature? The percentage figures gathered from the literature may decide whether in general the operation is tolerated. Say for instance that in every ten sinus operations in optic nerve disease of unknown origin we find in one a sinus disease and in one that the sight is considerably increased, then we have to ask ourselves whether we have the right to operate on eight patients unnecessarily to help two. The personal experience of both specialists is of influence too, for the experience of the ophthalmologist shows whether he has good clinical sense in choosing the patients on whom he allows an operation, while the experience of the rhinologist shows whether he has had good luck as an operator or not.

Knowing the possible advantage and possible damage the consultants will then have to decide whether in a special case they will operate or not and in this respect the personality of the patient will have to be considered, for we must always keep in mind that for the patient a sinus operation is not an indifferent procedure.

Though the nose can be made tolerably anæsthetic every

patient thinks a sinus operation very disagreeable and for a nervous patient it is a terrible shock, so that we cannot deny the influence on the psyche, especially when the operation has no effect. In strong-minded people we can discuss as far as possible the question with the patient himself, in others with the family; one of them should know that it is not a question of an operation for an existing disease but an explorative operation for a possible disease, otherwise they will always think, when no effect is gained, that the operator failed to do his duty.

Acting in this way I have not met a rhinologist who refused to do a sinus operation when requested and I have been very lucky in my results, so lucky that I hope I will always have the same fortune. These considerations show that we want statistics on the results of the operation both as to the restoration of vision and as to the results of damage for the nose and statistics of what happens with the eyes of patients in whom the sinus is not opened. To get these statistics it is necessary that we all use the same terms; up to now I am not convinced that this is the case. We see that in Nauheim von Eicken compares a statistic from Heine in Kiel who found in 50,000 patients 46 cases of retrobulbar neuritis (0.092%) and among them only 3 cases of nasal origin (0.006%), and a statistic from Grosz in Budapest, who found in 18,587 patients 58 cases of optic nerve disease of nasal origin (0.312%) or 52 times as frequent. Eicken suggests that this considerable difference may be caused by the fact that Grosz perhaps regards every case to be of nasal origin in which the sight is increased by an endonasal operation. If this is true it still must be explained how it is possible for Grosz to have 3.5 times more optic nerve disease of nasal origin than Heine retrobulbar neuritis of all origins. Either the optic nerve disease must be more frequent in Budapest than in Kiel or Grosz and Heine use the same name for different affections.

I think that it is very important to have a uniform nomenclature, and to try to get statistics of the advantages which the eye derives from a sinus operation and of the damage which is done to the nose.

In conclusion may I request your help in getting statistics to solve the question, should we operate on the nasal sinuses in optic nerve disease of unknown origin?

PERSISTENT ACCOMMODATIVE SPASM DUE TO LATENT HYPERPHORIA.

By Dr. F. W. MARLOW, Syracuse, N. Y.

(With one figure in the text.)

WHY some people accept readily a full correction of their hypermetropia and astigmatism as determined under cycloplegia, and some do not, is a problem the solution of which is often unsolved.

De Schweinitz states the problem thus:

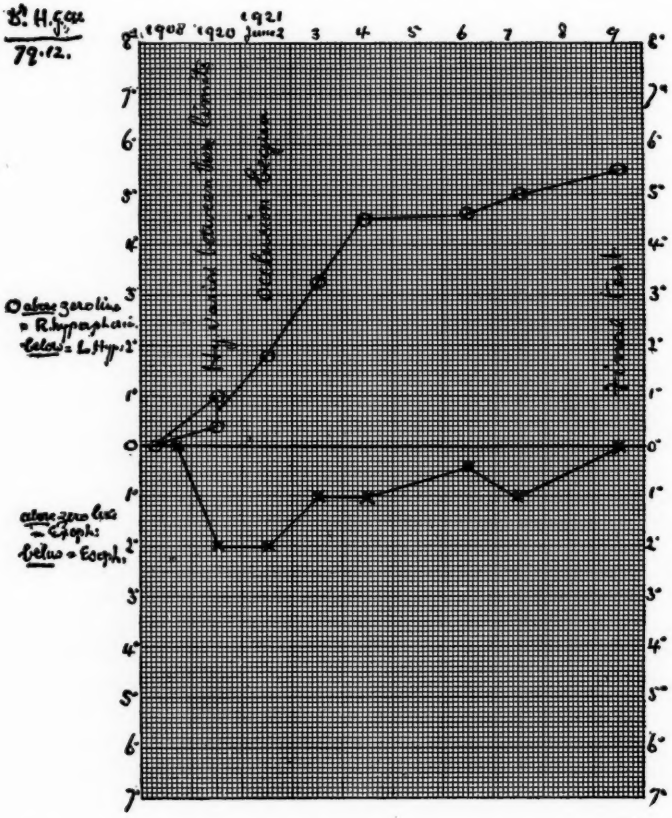
"After the effects of the drug have disappeared distant vision is often dim with the full correction, and a haze seems to lie over all distant objects, which disappears when the glasses are removed.

"Spasm of accommodation is the disturbing factor in this problem, and it is so variable in different individuals, that no precise rule can be given. Many persons wear a full correction with comfort, and do not need any modification, others will tolerate only a small part of the full correcting glass."

My own experience would lead me to modify slightly the last sentence and to say that a large percentage accept a full correction (−0.25D.) and a small percentage only do not. What are the factors in this small percentage which tend to maintain this accommodative spasm? The key to the solution of some problems of this kind is to be found in another paragraph of de Schweinitz—which says:

"A frequent cause of inability to wear a full correction depends upon the development of convergence insufficiency, causing an associated action of accommodation with the muscular action necessary to bring the visual axes in a parallel condition."

While it is not obvious how a convergence insufficiency would be operative in distant vision, it is easy to see how a divergence excess would have precisely the effect described in the latter half of the paragraph, and that any one of a number



of muscle anomalies may operate in just this way. In other words any excessive muscular effort necessary to bring the visual axes into parallelism may induce associated accommodative effort in excess of that necessary to maintain clear vision.

I have observed in more than one case in which at the post-cycloplegic examination the patient would not accept the full correction, that the placing of prisms in the trial frame to correct, wholly or in part, a manifest heterophoria will at once raise the vision or permit the acceptance of a stronger lens.

But in many cases there is no demonstrable heterophoria, or the correction of what heterophoria can be demonstrated does not bring about the desired result.

The following case, in which a high degree of hyperphoria was revealed by the prolonged occlusion test, and its correction followed by relief from symptoms suggests a possible solution of many other unexplained cases.

H. G. W., a medical student, seen first on Nov. 10, 1908, when he was 24 years old. He was complaining of his eyes getting inflamed, some aching, and other very slight asthenopic symptoms.

Examination showed: V. = R. $\frac{8}{8}$ −, + 0.37 S ⌒ + 0.37 C 110° $\frac{8}{8}$ +; L. $\frac{8}{8}$ − 0.25 S ⌒ + 0.62 C 85° $\frac{8}{8}$ − orthophoria.

After scopolamin: R. $\frac{6}{60}$ + 1.00 S ⌒ + 0.75 C 95° $\frac{8}{8}$ −; L. $\frac{6}{60}$ + 1.00 S ⌒ + 0.87 C 85° $\frac{8}{8}$.

At a post-cycloplegic test: R. accepted −0.25 S ⌒ +0.62 C 95°; L. accepted −0.25 S ⌒ + 0.62 C 85°.

He was ordered: R. + 0.50 Sph. ⌒ + 0.50 C 95°; L. + 0.50 Sph. ⌒ + 0.62 C 85°.

He received no benefit from the glasses, which also blurred distant objects, and after a month or a longer time gave them up.

In 1911 he was re-examined: R. accepted − 0.25 S ⌒ +0.50 C 85°; L. accepted −0.25 S ⌒ + 0.87 C 95°, right hyperphoria ½°.

After scopolamin: R. $\frac{6}{60}$ + 1.00 S ⌒ + 0.87 C 95°; L. $\frac{6}{60}$ + 0.75 S ⌒ +1.12 C 90°; right hyperphoria 1°.

At a post-cycloplegic examination seven days later: R. accepted − 0.25 S ⌒ + 0.75 C 95°; L. accepted − 0.25 S ⌒ + 0.87 C 85°, right hyperphoria 1°.

He was ordered: R. + 0.50 S ⌒ + 0.75 C 95°, ¾° prism base down, L. + 0.25 S ⌒ + 0.87 C 85,° with the same result as before. After a persistent trial of some months he gave them up and went without glasses until April, 1920, when he came again on account of recent inflammation of eyelids and general exhaustion after prolonged reading.

R. now accepted + 0.25 Sph. ⌒ + 0.37 C 95°; L. now accepted − 0.25 S. ⌒ + 0.62 C 75°; R. hyperphoria 1° esophoria 2°.

After scopolamin: R. accepted + 1.25 S ⊂ + 0.75 C 90°; L. accepted + 1.00 S ⊂ + 1.00 C 80°; R. hyperphoria ½°.

He was ordered: R. 1.00 S ⊂ + 0.62 C 90° ⊂ ½° prism base down; L. 0.75 S ⊂ + 0.87 C 80°.

On June 1st he returned complaining of blur and discomfort, vision being $\frac{6}{18}$ with the glasses, brought up to $\frac{6}{6}$ with the addition of −0.50 Sph. His spherical correction was reduced 0.25 Sph. in the hope that his accommodation would relax enough to accept the remaining correction, but a year later, June 2, 1921, having worn the glasses constantly in the interval, he still complained of blur for distance, disinclination to read, and fatigue afterwards.

R accepted + 0.25 Sph. ⊂ + 0.37 C 75°; L. accepted + 0.62 C 80°, or about the same as at his first examination on November 10, 1908. In other words the accommodative spasm remained unchanged.

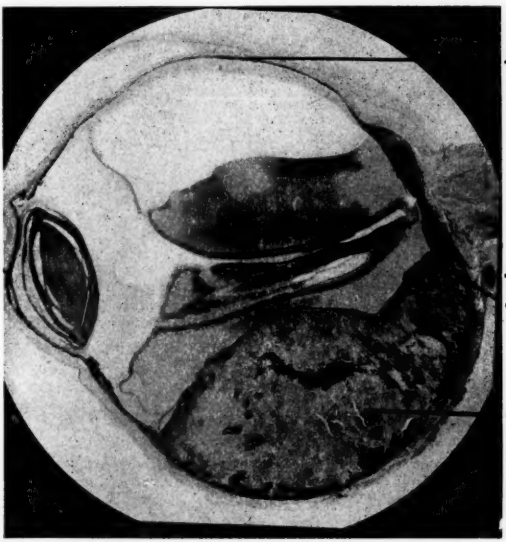
Test of the muscle balance now showed R. hyperphoria 1¾° esophoria 2°; p.p.c. 5*cm* from base line; abd. 3° − 5°, exophoria at ⅓ *m* with his glasses 3° − 4° prism; p.p. with his glasses, normal.

It had become evident that the simple correction of his refraction, full or partial, continued over a long period of time, was having no effect upon the accommodative spasm, that the latter could not be attributed to insufficiency of convergence, for that was above the normal standard, that moreover the correction of what manifest hyperphoria could be detected had no influence in relieving the symptoms, and the question seemed to arise naturally as to whether there might not be a higher degree of heterophoria present than could be demonstrated, which might on account of its necessitating increased innervation of extrinsic muscles induce also an excessive accommodative effort. In order to test this one eye was occluded with a ground glass and the effect watched. The test lasted seven days and the result is shown in the accompanying chart. The right hyperphoria increased until on the seventh day it reached 5½°. The esophoria diminished until on the seventh day it disappeared entirely.

He was ordered: R. + 0.75 Sph. ⊂ 0.62 C 85°, 2° prism base down; L. + 0.50 Sph. ⊂ + 0.75 C 80°, 1½° prism base up. This correction gave prompt relief from symptoms and clear distant vision, and he has remained comfortable since.

ILLUSTRATING DR. J. A. MACMILLAN'S ARTICLE ON "A CASE OF METASTATIC CARCINOMA OF THE CHOROID."

A. Tumor of choroid.
B. Point where cells have broken through Bruch's membrane.
C. Cancer involvement of retina.
D. Cancer involvement of optic nerve.



A. Large tumor.
B. Posterior ciliary vessels with perivascular involvement.
C. Extraocular nodule.
D. Small tumor in choroid on nasal side.

A CASE OF METASTATIC CARCINOMA OF THE CHOROID.

By Dr. J. A. MACMILLAN, Montreal.

From the Ophthalmic Clinic of the Royal Victoria Hospital.

(*With two illustrations on Text-Plate XIII.*)

CASES of metastatic carcinoma involving the choroid are still sufficiently rare to justify the presentation of another. Steichele (1) fully covering the literature up to 1918, could find only 71 reports of the condition. But in itself the following case presents special changes and problems that are of interest.

Mrs. J. P., age 46 years, secretary, was admitted to the Royal Victoria Hospital, in July, 1920, presenting all the signs of an advanced carcinomatosis, which had followed a mammary carcinoma of the left side notwithstanding a radical operation in February, 1919. Three months previously the sight of the left eye had disappeared suddenly during the night in association with severe pain.

In the family history it was to be noted that the maternal grandmother had died from cancer of the breast; a maternal aunt from cancer of the stomach; and that one sister had had her breast removed for cancer.

The left eye was not injected, except for a few dilated long ciliary veins on the temporal side. The cornea was clear, there was no anterior chamber, and the left pupil was slightly dilated. One could see a mass of a dirty yellow color, in the vitreous, on the temporal side. The retina appeared detached in all directions. Tension was 40 Hg. *mm* with the Schiotz tonometer. On transillumination there was a definite shadow with the light placed on the temporal side. Vision was nil. A diagnosis of intraocular tumor secondary to the mammary cancer was made; and it is interesting to note that the proper conception of this

case, in which the general findings were unconvincing, grew out of the ocular examination. The woman died on August 1, 1920. The autopsy report showed metastases of the lungs, liver, kidneys, adrenals, pancreas, pituitary, dura mater, and general involvement of the lymph glands. For the sake of completeness I will say here that the sections from these organs show the same cellular structure as that of the tumor in the choroid about to be described.

PATHOLOGICAL EXAMINATION OF THE EYE.

The left eye was hardened in formalin and alcohol, and embedded in celloidin. Sections were stained by hæmatoxylin and eosin, and by Van Gieson's method. On excising the upper and lower segments, before embedding, the whole temporal side of the eye was found lined by a grayish-white putty-like tumor, the thickness of which varied, being greatest near the equator. It was soft in consistency, and its surface was irregular. No hæmorrhages could be seen. The retina was completely detached, the sub-retinal space being entirely filled with fluid.

Examining sections macroscopically one notes the absence of an anterior chamber; the lens pushed forward; and the sclera everywhere intact. The retina is completely detached, except at the nerve-head and the ora serrata on each side. On the temporal side one sees a large mass which occupies somewhat more than one third of the vitreous chamber; it extends from the ora serrata on the temporal side to 4*mm* beyond the optic disk on the nasal side; and reaches its greatest thickness (8*mm*) near the equator. Throughout the mass there are areas which take the hæmatoxylin stain deeply. These are especially marked in the periphery, and stand out in sharp contrast to the eosin-stained central portion of the tumor. Similar strong affinity for hæmatoxylin is noted, along the temporal side of the nerve; in two minute spots, 1*mm* in size, outside the sclera at the posterior pole; and in a small patch (3*mm*) which is apparently situated in the choroid on the nasal side, 6*mm* distant from the nearest border of the large tumor. Everywhere beneath the detached retina the eosin stain is intense.

Microscopically the cornea is normal. The iris, thin and atrophic, with blood vessels of reduced caliber, is in close apposition to the posterior surface of the cornea. The posterior pigment layer is adherent in places to the lens capsule.

One sees the ligamentum pectinatum compressed, and Fontana's spaces obliterated. Schlemm's canal is small, and in some sections contains blood cells. The ciliary body, though flat, and with no exudate on its surface, shows many dilated and engorged blood vessels. The lens is pushed forward and in apposition with the iris. Lying against the stretched zonule on each side is a clear, highly refractile mass which, in all probability, represents the remains of the vitreous.

The tumor is made up of closely packed epithelial cells with deep-staining oval nuclei and very little cytoplasm. The nuclei are rich in coarse deep-staining chromatic granules, and show well-defined nucleoli. There is no glandular arrangement of the cells, and there is no stroma. Cells in all stages of degeneration can be seen; in fact the necrosis occupies the major part of the tumor. It is only at the margins, along the sclera, and around many blood vessels, that one finds the active cells which take the hæmatoxylin stain, and stand out as blue islands in the pink background of necrotic cells, as described above in the macroscopic examination. The blood vessels vary in size. Some are filled with blood, while others show an early obliterating endarteritis. One cannot definitely make out cancer cells in any of them.

The anterior margin of the tumor, on the temporal side, is wedge shaped, and extends to the beginning of the flat portion of the ciliary body, the cells having advanced along the lymph spaces of the choroid and separated its lamellæ. The nuclei here stain deeply. On the nasal side the advancing edge has the same appearance; but there is more dilatation of the choroidal vessels. The innermost part of the tumor is necrotic as I have already stated, and the only active cells are those immediately surrounding isolated blood vessels. Bruch's membrane appears everywhere intact except at a small spot near the disk, on the temporal side. Here the tumor cells seem to be continuous with the cells of another mass, which invades the retina and optic nerve. The choroidal pigment is found scattered in clumps throughout the tumor; but apart from this, there is nothing in the mass recognizable as choroidal tissue.

On the nasal side, corresponding to the dark area noticed macroscopically, is a growth 3.5*mm* long, the cells of which

are of the same character and invade the choroid in the same manner as those of the advancing margin of the large tumor. From its size and the staining of its nuclei, one would think the growth to be of more recent origin.

Invasion of the sclera is seen in many places adjacent to the large tumor. The cells have penetrated along its lymph spaces and separated many of its layers, the innermost of which are turned in, and appear as stroma for the overlying part of the tumor. At no place can a perforation be found.

The two dark stained nodules seen on the posterior surface of the sclera, macroscopically, are cancer cell nests, surrounded by connective tissue capsules. In some sections they are joined together and appear as one larger mass. There are no broken down cells, and the nuclei stain deeply. No direct communication with the intraocular growth can be found, but in some sections the cancer cells extend from the intraocular growth along the short posterior ciliary vessels for half the distance through the sclera.

Immediately to the temporal side of the disk the tumor cells appear to have broken through Bruch's membrane, and to have involved the retina. This involvement extends forward for a short distance (*1mm*) only, but well backward through the lamina cribrosa into the substance of the optic nerve (*2mm*). The pigment epithelium has remained attached to Bruch's membrane throughout, but the retina otherwise shows complete disintegration. The central vessels of the optic nerve are not involved, the reason being that the growth does not extend so far as to the nasal side. On the temporal side, however, its involvement is up to the pial sheath.

From the character and manner of growth of the epithelial cells, together with the absence of glandular arrangement and stroma, one could only make a diagnosis of medullary carcinoma.

REMARKS.

It will be seen in this case that we have three metastatic growths associated with the eye: the large one on the temporal side, a small one on the nasal side, and the extraocular nest lying on the sclera. It would appear from the size and the extensive necrosis that the original metastatic growth

was the one on the temporal side, and that it had penetrated Bruch's membrane near the nerve head, involving the retina, and extending from there directly along the lymph spaces into the optic nerve. The extraocular nodule is probably secondary to the large tumor, because it lies directly over the area where the short posterior ciliary arteries penetrate; and in some sections vessels with perivascular involvement can be found extending from the intraocular tumor, halfway through the sclera. Had serial sections been cut it is extremely probable that this perivascular involvement would have been found to lead to these nests. There is no involvement of the choroid between the nasal nodule and the large growth, so that the former must have had its origin by metastases through the blood stream, but from what focus, it is impossible to state. One would like to consider it as secondary to the temporal tumor; but it is difficult to assume a vascular connection. On the other hand, with general carcinomatosis, the blood stream frequently must have contained clumps of cancer cells, and I would incline to the view that one had to deal here with multiple foci.

As the original growth in this case was on the temporal side, posteriorly, it might be well to reiterate the accepted view as to the cause of the frequency in this location. It is first of all necessary to believe that metastases occur by embolism through the blood stream, as one cannot imagine the extension from the breast to the eye by the lymphatics alone. The ophthalmic artery leaves the carotid at a right angle, which probably explains the infrequency of ocular metastases. In its further course through the orbit the short posterior ciliary arteries going to the temporal side come off usually as one large branch, the axis of which is parallel to the long axis of the ophthalmic. This branching occurs at the point where the ophthalmic artery changes its course upward, so that an embolus, once in the ophthalmic, is more likely to be directed to the temporal side, along the short posterior ciliary artery.

This case yields no evidence of the view that the detachment of the retina is out of proportion to the size of the tumor in metastatic carcinoma of the choroid. Oatman (2) believes that there is an early effusion from the tumor itself to explain in part the relatively large separation. In this case, one has had

bulk and interference of the circulation as more likely causes.

Marshall (3) noticed that the tension in carcinomatous eyes was usually lower than in cases of sarcoma. This he attributed to a peculiarity of metastatic carcinomata, which are usually thin and flat, and consequently less likely to push forward the lens and iris and to block the angle of the anterior chamber. In this case there was a tension of 40 Hg.*mm* (Schiotz) which, although elevated is surprisingly low when one considers the large size of the tumor, and the fact that the lens was pushed forward, completely blocking the angle. The findings here seem to agree with Marshall's, in that the tension was comparatively low; but his explanation certainly does not apply to this particular case. It will be necessary to have tonometer readings on future cases to definitely confirm his findings, as the digital method was used in all the cases collected by him. An interesting feature in the sections of this case was the small amount of vitreous found, and the questions arise, if the absorption of the vitreous was compensatory to the growth of the tumor, and if this had some effect in keeping down the tension.

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INTERMITTENT OPHTHALMOMALACIA.

By Dr. S. O. FIELDS, Norfolk, Va.

WE reached the decision to report the following case, not alone because the condition is infrequently met with, but also because of certain noteworthy characteristics which seemed to merit especial attention. The case furthermore exhibits features which may prove of interest to the internist and the laryngologist.

L. D., colored, female, age 30 yrs., rural school teacher, came under my notice, Aug. 1, 1921, in the Eye and Throat Department of the Princess Anne Avenue Clinic, complaining of recurrent attacks of tonsillitis and also of occasional attacks of impaired hearing. In addition she stated that her friends had begun to comment upon the increasing circumference of her neck, but that she herself had paid no attention to it believing it simply to be an accompaniment of her increasing stoutness, although she admitted she now and then felt a sensation of moderate constriction about the throat. She also during her recital volunteered the information that her right eye had been slightly painful and sensitive to the sunlight for the last two or three days, and that such attacks occurred at irregular intervals of never more than three or four weeks in length, the attacks lasting sometimes two or three days and sometimes a week. She stated that very often when rubbing her eye at these times she noticed the resemblance to a "soft rubber ball partly filled with water." She has an entire absence of any other symptoms, and only when asked a leading question does she recall that she is annoyed now and then with palpitation of the heart.

There is nothing of importance in the family history, and also nothing in the past medical history save the frequent attacks of tonsillitis and the occasional slight hardness of hearing.

The patient is a stout, rather intelligent young woman. General examination shows an unequal bilateral enlargement of the thyroid, the enlargement being decidedly more marked on the right side; no thrill or pulsation in the gland; circumference of the neck 16½ inches. Heart rate, 128; forcible action; no murmurs; second aortic sound accentuated; slight increase in the cardiac area. Tremor of the outstretched fingers. Laboratory examination gives us no data of interest. Systolic pressure 130; diastolic, 100*mm.*

Nose, normal; throat, tonsils moderately enlarged, and about 2*cc* of pus squeezed out of the two tonsils.

Each eye exhibits about 25*mm* of exophthalmos, and the palpebral fissures are increased in width, the right, however, to a much slighter extent than the left. Both eyes show Stellwag's sign, von Graefe's sign, tremor of the upper lids when gently closed, and an absence of Moebius' sign. No injection in either eye. While the left eye exhibits a normal pupil and normal tension, the right eye on the other hand shows a pupil which does not dilate when shaded and a minus 2 tension, the latter allowing us easily to wrinkle and indent the cornea by pressure with a probe. The 2% cocain solution employed to permit the use of the probe on the cornea produced no dilatation of the right pupil. She wears a plus 0.75 D. Cyl., axis 90 degrees before each eye and a refraction done in the clinic verified the fact that the correction was correct. There is nothing else necessitating especial mention from the ocular standpoint, no eye lesions being discovered to account for the diminished tension.

She was given medicinal treatment and advice by the General Medical Department, and advised to submit to a tonsillectomy when her cardiac symptoms should have become somewhat ameliorated. She entered the clinic again Sept. 2, 1921, with her ocular tension normal and equal in both eyes and reported an attack of her usual eye trouble about two weeks before. A tonsillectomy was done under local anæsthesia with uneventful recovery.

She was seen again Oct. 26, 1921, while visiting the city. The circumference of the thyroid had decreased to 13½ inches; her heart rate was 90 per minute; no tremor of the outstretched hands. Her exophthalmos is still the same; the right palpebral fissure is now of the same width as the left; the right pupil dilates when shaded; the cornea cannot be indented or wrinkled by the probe. She states that she has had no photophobia or ocular pain since the tonsillectomy. Although ordered to report regularly the state of her gland and the condition of her eye the patient left town to teach and we have since lost trace of her.

Here then, briefly, we have a thyrotoxic patient, a sufferer from infected tonsils, who exhibits an irregular periodic softening of the eyeball on the side of the greatest degree of glandular enlargement, who reveals no other cause for the intermittent minus tension that can be demonstrated anatomically or clinically, and who not only reports a marked subsidence of the thyrotoxic symptoms after tonsillectomy, but also the disappearance of the periodic attacks of ocular trouble.

Therefore we designate this case—and we believe with much justification,—as one of intermittent ophthalmomalacia. The term is to be restricted to that condition characterized by periodic softening of the eye, in which we can find no ætiologic factor in the ball itself, such as retinal detachment, phthisis following trauma, iridocyclitis, or panophthalmitis. At intervals the eye becomes markedly soft, with a little injection, a little photophobia, a little pain, perhaps, but the symptoms are not marked and the condition soon subsides, the vision remaining as good as ever. Intermittent ophthalmomalacia was first described by von Graefe, and the causes are in most instances as obscure and unknown as they were then. Most authorities dismiss the subject with the statement that injuries of the neck, tumors of the upper part of the chest or posterior mediastinum, or enlarged thyroid or lymphatic glands are probable causal factors in some instances. In the majority of cases of intermittent ophthalmomalacia where the cause is known, the ætiologic factor has been found to be an enlarged thyroid which becomes swollen from time to time and so produces a pressure paresis of the sympathetic nerve on the side affected, the effects of the paresis disappearing with the subsidence of the extra amount of swelling.

Clinical as well as experimental evidence has given us a fairly definite train of ocular symptoms as indicative of sympathetic nerve paresis. The ptosis of the upper lid due to the paresis of the superior palpebral muscle which is supplied by the sympathetic; the contraction of the pupil because the paretic dilatator allows the sphincter muscle to preponderate; the sinking in of the eyeball from paresis of Mueller's orbital muscle which normally aids in preventing recession of the globe; the decrease of ocular tension sometimes found accompanying the foregoing signs: all these phenomena we now know to be

definitely characteristic of sympathetic nerve paresis. In our case two ocular features are worthy of attention as having a probable bearing on the cause of the exophthalmos and the widening of the palpebral fissure found in hyperthyroidism. Landström and others dispute the theory that the exophthalmos in Basedow's disease is due to a dilatation of the orbital vessels and prefer to ascribe it to a tonic contraction of the smooth fibers of the orbital muscle which are supplied by the sympathetic. In our case, however, the sympathetic was paretic and yet the exophthalmos was not less in the right eye than in the left, this fact making it appear, therefore, extremely unlikely that the exophthalmos is due to tonic contraction of the orbital muscle to any marked extent—if at all. Apropos of the gaping of the palpebral fissure found in hyperthyroidism we have had a number of theories offered, the foremost being the theory of increased tonicity of the levator advanced by Moebius and the theory of tonic contraction of the smooth superior tarsal muscle supplied by the sympathetic. It is probable that both are important factors in the production of the gaping palpebral fissure, for in our case in which the sympathetic exhibited the signs of intermittent pressure paresis, although we found the right fissure less wide than the left still the gaping was too great in extent to be accounted for by the proptosis alone, and so Moebius' increased tonic levator contraction was probably also present.

The consideration of the chain of events in this case brings us face to face with the following facts. The favorable effect exerted by the tonsillectomy on the whole process indicates the probability of the tonsils being at the bottom of the whole trouble. We believe the tonsils acted as infective foci to produce a perverted condition of the thyroid gland, this grandular involvement being attended by characteristic enlargement and both general and ocular symptoms. The attacks of intermitting ophthalmomalacia were evidently brought on by the periodic swelling of the large thyroid, the eye being affected on the side with the greater thyroid enlargement because it was on that side only that the periodic increase in size permitted pressure on the sympathetic.

THE SIGNIFICANCE OF MOLLUSCUM CONTAGIOSUM AS AN ÆTIOLOGICAL FACTOR OF CONJUNCTIVAL AND CORNEAL DISEASE.

By Prof. ANTON ELSCHNIG, Prag, Czecho-Slovakia.

RECENTLY H. and S. R. Gifford reported six cases in which mollusca contagiosa located at the lid border were proven to be the ætiological factor of a long-standing chronic conjunctivitis. Reference was made to the few reports that had appeared in the previous literature. In reality, the ætiological significance of molluscum contagiosum in cases of conjunctivitis has been known but a comparatively short time, and to de Wecker belongs the credit of having recognized it first. In 1897, I published seven cases, one of whom I had had under observation for four years. In six of these cases there was a chronic conjunctivitis, with multiple follicle formation that so closely resembled a trachoma that the diagnosis of trachoma had been made elsewhere and treatment for that condition begun. The seventh was a simple chronic conjunctivitis. In all of these the diagnosis of molluscum contagiosum was confirmed by the anatomical examination and in all of them, the conjunctivitis disappeared following the extirpation of the molluscum. At that time, I expressed the opinion that the influence of the molluscum upon the conjunctiva was partially a reflex, and partially a mechanical irritation due to extruded molluscum cells. In support of the first view may be mentioned the rather common observation that frequently a chronic conjunctivitis is continued by the presence of small papillomata at the roots of the cilia and can be relieved only by the removal of such. The second view is confirmed by the undoubted infectious character of the molluscum.

In a later communication, I was able to show that molluscum of the lid margin may lead not only to a conjunctivitis, either with or without a trachoma-like follicular hypertrophy, but also to a conjunctivitis or even a keratitis of a phlyctenular or eczematous character. The latter is merely further proof that an irritation, which in a normal individual would be followed simply by a conjunctival inflammation, may cause a typical phlyctenular affair in an individual so predisposed (exudative diathesis). A diagnostic tuberculin injection may be followed by an acute phlyctenular disease in the same manner, just exactly as a simple inflammation of the conjunctiva due to pneumococcus or Koch-Weeks bacillus, may cause the outbreak of a phlyctenular kerato-conjunctivitis in the individual so predisposed. In those cases, the removal of the molluscum is essential for the relief of the phlyctenular disease.

In the Clinic we see about ten thousand cases a year and among these, there will be four cases of molluscum contagiosum so that in each semester it is possible to demonstrate one or two cases.

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PRIMARY INTRANEURAL TUMORS (GLIOMAS) OF THE OPTIC NERVE.

A HISTOLOGIC STUDY OF ELEVEN CASES, INCLUDING A CASE SHOWING CYSTIC INVOLVEMENT OF THE OPTIC DISK, WITH DEMONSTRATION OF THE ORIGIN OF CYTOID BODIES OF THE RETINA AND CAVERNOUS ATROPHY OF THE OPTIC NERVE.[1]

By Dr. F. H. VERHOEFF, Boston, Mass.

(Concluded from March number.)

COMMENT.

Case 1 shows one feature which has not hitherto been observed, namely gliomatous involvement of the optic disk and retina with formation of prepapillary cysts. Contrary to what might have been expected, the involvement of the disk was not due to direct extension of the tumor from the nerve stem through the lamina cribrosa, for, as a matter of fact, the portion of the nerve within and just behind the lamina was of almost normal thickness, but obviously to proliferation of the preëxisting neuroglia of the disk. The occurrence of cysts in such tumors has been repeatedly observed and the cysts on the disk in this case are unique, therefore, only as regards their situation.

From a clinical standpoint also the case is unusual in that the larger cyst of the disk was recognized as such on ophthalmoscopic examination, and in that there was a definite record of normal vision in the eye, antedating the onset of exophthalmos for years. At the first ophthalmoscopic examination, the

[1] From the Massachusetts Charitable Eye and Ear Infirmary.

appearance was that of ordinary papillœdema. This later became more marked and then apparently subsided somewhat. Two years elapsed previous to the last ophthalmoscopic examination so that the development of the cyst could not be followed. Whether or not the appearance of papillœdema was from the first due to gliomatous involvement of the disk it is impossible to determine.

Gliomatous involvement of the disk apparently also existed in Case 2, but in an early stage.

In the literature I have found only one case of glioma of the optic nerve, that reported by Sulzer and Rochon-Duvigneau,[1] in which the optic disk possibly showed gliomatous involvement. In this case a tumor of the disk was seen ophthalmoscopically and taken to be a cyst; but on microscopic examination the disk was found to be replaced by a solid vascular growth. The authors do not state that the latter was composed of neuroglia, and their description of it is too brief and inadequate for any definite conclusion to be drawn as to its nature.

Case 4 is especially noteworthy. In this case, four years after removal by Dr. de Schweinitz of a tumor of the orbital portion of the optic nerve, which I find to be a typical glioma, symptoms of a tumor of the hypophysis developed, and at a second operation a tumor of this body was actually found and partly removed. Sections of the hypophysial tumor which I examined, show it to be a typical glioma arising from cells of the pars nervosa. A possible relationship between the two tumors is suggested below. Case 10 is unusual in the occurrence within the tumor of numerous cells resembling ganglion cells. These cells were regarded as true ganglion cells by Dr. Ewing and Dr. Meeker, but for reasons given in my description of the case, I regard them as neuroglia cells. In Case 7 the tumor was unusually large and contained unusually large cysts, markedly dilated veins, some of which contained fibrinous thrombi, large interstitial hemorrhages, and an unusual abundance of cytoid bodies.

The tumor in Case 9 presents one feature not present in any of the others, namely, a large area of hyaline necrosis

[1] Sulzer et Rochon-Duvigneau: "Neoplasme du nerf optique et de la papille," *Ann. d'ocul.* cxlix., 161, 1913.

containing numerous corpora amylacea. In the vicinity of this area are many endothelial phagocytes, some of which have formed foreign body giant cells.

All of these tumors are sufficiently similar as to leave no doubt that they are all of the same nature, yet no two of them are exactly alike in structure. The differences between them are chiefly quantitative, that is, generally speaking, the same elements can be seen in all of the tumors, but in different proportions and arrangements. That they are all composed primarily of neuroglia is sufficiently obvious even without the use of differential stains. When a portion of the normal or relatively normal nerve is present in the specimen, the transition of the normal neuroglia into that of the tumor proper is apparent at a glance. The results, however, obtained by the use of Mallory's neuroglia stain in five cases (Cases 1, 2, 6, 9, and 10), in which the tumors included all the different types of tissue encountered in the other tumors, leaves no doubt as to the gliomatous nature of all these tumors. In the other cases the fixation was unsuitable for the use of the neuroglia stain.

While the histologic structure of most of these tumors at first appears rather complicated, yet it seems to me possible to recognize three main types of tissue in them. Any one type may undergo transition into either or both of the others. The first which, for lack of a better term, I shall designate the finely reticulated type, is similar to, but not identical with, that of the neuroglia of a normal optic nerve (Figs. 7 and 8). It consists of a matrix of fine fibrils running in the interspaces of a delicate cell reticulum, embedded in which are small round or oval nuclei. The fibrils may run irregularly or all in the same general direction. The reticulum may be so delicate as to be scarcely recognizable, or it may be quite definite. The tissue is usually marked off by connective tissue septums into definite spaces as in the case of a normal nerve, but sometimes occurs in irregular and less well defined masses. The nuclei may be scanty or abundant, and regular or irregular in size. They are generally surrounded by only a small amount of cytoplasm, which stains feebly and generally contains small vacuoles. Occasionally cells occur, however, which have a considerable amount of cytoplasm, eccentrically placed nuclei, and long processes which give them considerable resemblance

to ganglion cells. Such cells are present in Cases 9 and 10, being especially abundant in the latter, and for reasons given in the description of these cases, I regard them as undoubtedly neuroglia cells.

The second type of tissue, which I shall designate the coarsely reticulated type, seems to be simply an exaggeration of the first type (Figs. 4 and 10). The nuclei are similar, but the reticulum of the syncytium much coarser, and vacuolization of the cytoplasm is extremely marked, producing appearances resembling those of a myxoma. Fibrils are coarser and fewer in number than in the first type. This tissue has a special tendency to form cystic spaces, often of large size. It is well exemplified by the extraneural portion of the tumor of Case 8, and it was no doubt upon the appearance of this tissue that Salzman based his diagnosis of myxosarcoma in this case.

The third type of tissue I shall designate the spindle-cell or coarsely fibrillated type. This consists of coarse neuroglia fibrils, many of which are partly spiral in form, between which lie neuroglia cells. The latter are, generally, so-called spindle cells, and these may be so abundant as to make the tissue closely resemble that of a spindle-cell sarcoma (Figs. 5 and 9). The cells are often similar to the spindle cells which occur in a normal optic disk just anterior to the lamina cribrosa and which Nikolai regarded as smooth muscle fibers.[1] Occasionally, most of the cells are similar to those of Type 1. The fibrils are generally arranged in fairly definite bundles. The cells form a vacuolated syncytium with lateral communications between which the fibrils run, so that when the bundles are seen in cross section they present an appearance more or less similar to the tissue of Type 2. When, therefore, the bundles run very irregularly so that many of them are seen in cross section, two types of tissue may appear to be present whereas this is not the case. Without special staining the longitudinal bundles might be, and no doubt often have been mistaken for, connective tissue stroma. When stained differentially, the neuroglia fibrils appear to be more abundant in this type of tissue than in the others, but this may be due to

[1] Verhoeff, F. H.: "Some Remarks on the Use of Mallory's Phosphotungstic Acid Hematoxylin, and a Note on the Musculus Papillæ Optici of Nikolai," *Roy. Lond. Ophth. Hosp. Rep.*, xv., 4, 1903.

their being more conspicuous. This type is well exemplified by the tumors in Cases 2 and 11, in which it was the only type of tissue present. In some cases, as in Case 9, in areas in which there is rather abrupt transition from Type 1 to Type 2, there is a marked tendency for the spindle cells to arrange themselves radially to the connective tissue septums. The spindle-cell type of tissue also tends to occur in close relation to the connective tissue stroma. When the tumor invades the latter, as it often does, or the pia and dura, the invading tissue is practically always of this type, and is often so intermingled with the connective tissue elements that without special staining its true nature may escape recognition. Case 10 is unusual in that the spindle-cell type of tissue occurs in the interseptal spaces, while the coarsely reticulated type occurs along and within the septums.

None of the types of tissue just described closely resemble the tissue of an embryonic optic nerve at any stage, but Type 1 somewhat resembles the syncytium of an embryonic optic disk at an early stage, while Type 3 is somewhat similar to the tissue of the embryonic nerve stem. The embryonic optic nerve at this stage, however, does not contain any fibrils which stain differentially in the neuroglia stain. Tissue, identical with that of Types 2 and 3, often results from hyperplasia of the neuroglia of separated retinæ which occur under inflammatory conditions.

In none of these tumors can I find any neuroglia nuclei in the process of either direct or indirect division, owing, no doubt, to the very slow rate of proliferation. In sections of the five tumors stained in Mallory's neuroglia stain, all of the tumor cells show centrosomes, each containing two dots, and no cells are to be found containing centrosomes with multiple dots such as are seen in ependymal cells and some of their derivatives. Occasionally, some of the tumor cells contain hematogenous pigment. In no case do the tumor cells tend to become free and infiltrate the tissues.

A feature of these cases deserving some comment is the proliferation of the arachnoid tissue which often takes place, especially at the anterior end of the subdural space. It is marked in Cases 1, 2, 3, 7, and 9. In the other cases the sections did not include this part of the nerve. The new tissue is

easily recognizable as arachnoid tissue, although it is often atypical. In places the stroma in the form of hyaline strands predominates, while in others the cellular elements do so. When this tissue is invaded by the tumor, as in Cases 1, 2, 3, and 7, there naturally results complicated appearances, which when seen in sections tangential to the nerve, might lead to a diagnosis of endothelioma. In Case 7 the arachnoid had produced large hyaline areas which had been surrounded and invaded by the tumor so as to produce appearances not unlike those of an epithelial cylindroma. The cause of this arachnoid proliferation I take to be purely mechanical, namely, the distention of the subdural space and the consequent strain of long duration thereby put upon the arachnoid trabecula. That such distention, if long continued, is sufficient to cause marked proliferation is almost certain, for I have observed it in cases of orbital tumors not connected with the nerve, which had produced papillœdema. The possibility that some stimulating influence from the glioma is a contributory factor is not absolutely excluded, but is unlikely from the fact that these tumors show no special tendency to stimulate other connective tissue even when in direct contact with it.

A question of special interest relates to the mode of origin and growth of these tumors. Most tumors occurring elsewhere evidently arise from a small focus and increase in size by continuity of growth, pushing aside or invading the contiguous tissues. In the case of the tumors under consideration, the growth does not increase in size by invading or destroying the original nerve structure, but by causing the preëxisting neuroglia in the vicinity of the growth to proliferate. In longitudinal sections which include a portion of the uninvolved nerve, the gradual transition of the normal neuroglia into that of the tumor is most obvious. In some cases, such as Case 1, it is only when the growth invades the subdural space, where it meets less resistance, that the neuroglia takes on a markedly abnormal character. It is noteworthy also that in this case, in which the retina was involved, the retinal hyperplasia involved only the preëxisting neuroglia and tended here to produce tissue of a relatively normal character. These facts seem to show clearly that either some abnormal stimulating factor or, less probably, the lack of some normal inhibitory

factor, has existed in the affected portion of the nerve and retina. Possibly such a stimulating factor is supplied in increasing abundance by or from the actively proliferating neuroglia of the tumor. This would best explain the proliferation of the neuroglia of the disk and contiguous retina in the case referred to, and perhaps also the glioma of the hypophysis in Case 4. It would also explain the notable fact that in the literature there is no record of a single case in which recurrence of a glioma of the optic nerve has taken place, although in many, if not most, of the cases the removal has been incomplete.

Since the preceding was written, I have found that Councilman[1] has come to similar conclusions in regard to gliomas of the brain. He states: "The advance of the tumor is preceded by a general proliferation of the neuroglia cells which become converted into cells of the tumor and have the growth and form characteristic of these. . . . It is impossible to avoid the impression that in the tumor, or in some way associated with it, a substance is produced which stimulates the neuroglia to growth; this growth taking the form both of general gliosis and of tumor."

The fact that the great majority of these tumors of the optic nerve manifest themselves early in life suggests that they are really congenital in origin and due to some more or less localized abnormality in the embryonic development of the neuroglia of the nerve. A tumor having once begun from such a congenital "anlage" might be partly or wholly dependent for its further development upon the stimulating factor just hypothecated. This possibility is not excluded by the fact that in Case 1 there was normal vision in the affected eye at the age of 10 years, since it is not necessary to assume any defect in the formation of the optic nerve fibers.

As already mentioned, Hudson did not regard these tumors as true gliomas, but preferred the designation "degenerative gliomatosis, implying a generalized overgrowth of neuroglia tissue of infiltrative character, dependent on some degenerative change in the tissues of unknown ætiology." He apparently based his view entirely on the fact that they do not recur after

[1] Councilman, W. T.: "Anatomical Consideration of Tumors of the Brain with Special Reference to the Gliomata," *Colorado Med.*, xii., 289, Oct., 1915.

incomplete removal. Since we do not know the cause of gliomas of the brain, this term does not necessarily indicate any essential difference between many gliomas of the brain and these tumors of the optic nerve and I therefore see no reason for its adoption. In my opinion, the facts that these tumors of the optic nerve are composed primarily and almost exclusively of neuroglia, that they invade the connective tissue stroma, penetrate the pia, and grow exuberantly and atypically in the subdural space, and are histologically similar to many gliomas of the brain and spinal cord, are alone sufficient grounds for regarding them as true gliomas.

While certain types of glioma of the brain are similar to gliomas of the optic nerve, other types frequently occur in the brain, which are markedly dissimilar to any of the optic nerve tumors. Such tumors of the brain contain elements which are more closely related to those of the embryonic neural epithelium, and are, perhaps, derived from persisting remnants of this epithelium. Since the optic nerve, so far as I know, never contains ependymal cells or remains of the embryonic neural canal, it is not surprising that the tumors arising from it never contain structures, such as rosettes, which suggest a derivation from these elements.[1] For a similar reason, tumors of the optic nerve would not be expected to contain ganglion cells.

Since these tumors of the optic nerve in their gross appearances bear considerable resemblance to neurofibromas of the peripheral nerves, it is not surprising that attempts have been made to prove that they were of the same nature or were dependent upon similar causes. Goldman,[2] who made the first attempt in this direction, and later Byers,[3] based their arguments chiefly on the assumption that the tumors of the optic nerve were composed primarily of mesoblastic tissue and were therefore histologically similar to neurofibromas. Now that

[1] Sulzer and Rochon-Duvigneau (*Ann. d'Ocul.*, cxlix., 161, 1913) mention epithelioid masses resembling rosettes as occurring in the tumor referred to above; but from the meager description given it is impossible to determine whether or not they were analogous to the rosettes occurring in tumors of the brain.

[2] Goldman, E. C.: "Beitrag zu der Lehre von den Neuromen," *Beitr. z. klin. Chir.*, x., 13, 1893.

[3] Byers: "The Primary Intradural Tumors of the Optic Nerve," *Studies from the Royal Victoria Hospital*, Montreal, 1901.

we know that the tumors are composed essentially of neuroglia, this argument falls to the ground.

The possibility still remains, however, that the same influence which causes the supporting tissue of a peripheral nerve to proliferate in the form of a fibroma, may cause the supporting tissue of the optic nerve to proliferate in the form of a glioma. This theory was suggested by Emanuel,[1] who, however, regarded most of these tumors as mesoblastic in origin. The only important evidence he brought forward, consisted of the facts that in one of his cases the father of the patient was affected with fibroma molluscum, that there was a history of the same condition affecting his grandfather, and that a brother was affected with pigmented nevi. If Emanuel's theory were correct, it would seem that among the numerous cases of glioma of the optic nerve that have been reported in the literature, in at least one case the two conditions should have coexisted. Moreover, as Hudson points out, "the existence of multiple isolated tumors of the optic nerve, which Emanuel adduces in support of his theory, has apparently never been conclusively demonstrated by microscopic examination."

VACUOLIZATION AND CYST FORMATION IN GLIOMAS OF THE OPTIC NERVE AND THEIR SIGNIFICANCE IN REGARD TO CAVERNOUS ATROPHY OF THE OPTIC NERVE.

Vacuolization is such a marked characteristic of neuroglia cells, and particularly of the cells in these tumors, that it is worthy of special consideration, although it is not, of course, peculiar to neuroglia cells. It is, for example, a striking characteristic of the cells of the notochord and of tumors arising therefrom. In all of the tumors of the optic nerve stem, which I examined, it was a well-marked feature, although more so in some than in others. In the tumors reported in the literature it evidently was equally as marked, although it was not recognized as such and was frequently mistaken for myxomatous degeneration. Thus in fifty-four out of the 118 cases collected by Hudson, the tumor was regarded as partly or wholly myox-

[1] Emanuel: "Ueber die Beziehungen der Sehnervengeschwülste zur Elephantiasis neuromatodes und über Schnervengliome," *Arch. f. Ophth.*, lxxii., 129, 1902.

matous in nature, and of these, thirty-five were reported as myxosarcomas. It is noteworthy, moreover, that not a single observer reported a positive test for mucin.

In studying sections of these tumors, I find that the process of vacuolization occurs in all types of neuroglia cells, and I have gathered the impression that it is upon this process that the formation of the delicate communications between neighboring cells depends, or in other words, that the reticulated syncytium characteristically produced by neuroglia cells, is dependent upon a process of vacuolization, the minute spaces between the trabeculæ representing vacuoles within the syncytium, and the larger spaces vacuoles which have broken into one another. I have also frequently observed identical appearances in the glia tissue formed by separated retinas under inflammatory conditions, and in diffuse gliomas of the brain.

I find also that the cysts which frequently occur in these tumors and also in diffuse gliomas of the brain are obviously due to a further extension of the process of cell vacuolization. The smaller spaces and vacuoles break into one another, and in this way form cysts of various sizes. This process is well shown in Figure 4, where the vacuoles in the surrounding cells can be seen in actual communication with the cyst cavity. It is conceivable, of course, that cysts of a different origin may occur in these tumors; but I have never encountered them.

The original contents of the vacuoles and spaces is a matter of conjecture. In the sections, most of the spaces are apparently empty, only a few, and these comparatively large, containing dilute serum. They were, therefore, originally filled with fluid which has diffused out in the preparation of the specimens. Since after fixation, no albuminous precipitate or coagulum has been left, the fluid may have been similar to cerebrospinal fluid. In Case 7, however, it is noteworthy that the trabeculæ around many of the spaces are coated with fine granules and threads, stained deeply in hematoxylin. I have seen similar granules on the trabeculæ around the cavernous spaces in optic nerves of glaucomatous eyes, and also on the trabeculæ around the cystoid spaces frequently found in the retina near the ora serrata in senile eyes. It is possible that these may consist of mucin; but even if this should be true, it would be improper, or at least misleading, to designate the

tissue myxomatous, since it is not mesoblastic in origin. The cystic cavities, and many of the larger spaces in the tumors, contain serous coagulum of various degrees of density, similar to the serous fluid which collects beneath separated retinas. This serum is evidently not derived from the cell vacuoles, but is exuded more directly from the blood vessels into the spaces and cysts. Some of the cystic spaces also contain red blood corpuscles. In Figure 4 it will be noted that while the cyst cavity proper is filled with serum the surrounding cell vacuoles are apparently empty.

By comparing sections of optic nerves showing cavernous atrophy with sections of these tumors, I find that in the latter the exact picture of a glaucomatous cup surrounded by cavernous spaces often occurs. On the trabeculæ around the cavernous spaces of the optic nerve, as noted before, I have occasionally also found basophilic granules as in Case 7. It is evident, therefore, that the cystic spaces occurring in cases of cavernous atrophy of the optic nerve are due to a natural tendency of neuroglia to produce cysts. In other words, they are due to hyperplasia of the neuroglia of the nerve and not primarily to degeneration, as has been assumed. I do not, however, concur in the view of Schnabel that the main glaucoma cup itself is formed by cavernous spaces breaking into one another, because the excavation of the disk frequently takes place before cavernous spaces in the nerve are produced.

THE ORIGIN OF CYTOID BODIES.

Another feature of these tumors requiring further consideration is the occurrence of cytoid bodies within them. These bodies undoubtedly have previously been seen by other observers, generally being described as hyaline masses occurring within or between the tumor cells, but their identity with the cytoid bodies occurring in the retina under various pathologic conditions has not hitherto been recognized. The latter have been regarded as varicose nerve fibers, degenerated ganglion cells, and degenerated neuroglia cells, by different observers. Since in these tumors the bodies present all the various appearances shown by cytoid bodies of the retina in albuminuric retinitis and other conditions, and since in both situations the

tissues in which the bodies mainly occur, namely, the nerve fiber layer of the retina, and tissue derived from the optic nerve stem, are similar in nature, there seems to be no escape from the conclusion that the bodies in the two situations are identical. As it has hitherto been impossible to ascertain the origin of cytoid bodies in the retina, owing, no doubt, to their scarcity in any one case, I have attempted to ascertain their origin in these tumors in which they are often abundant.[1]

At the outset, the possibility of their being varicose nerve fibers can be excluded by the fact that they are likely to be especially abundant in the subdural portion of the growths where nerve fibers have never existed, while an origin from ganglion cells is excluded by the fact that there are no definitely recognizable ganglion cells in the tumors.

As seen in these tumors, the cytoid bodies when small are often round, and rarely encapsulated. The larger ones are generally elongated and highly irregular in shape (Figs. 10 and 11), and sometimes roughly sausage shaped, seldom pyramidal. The largest may attain a cross diameter of .025*mm*, and they may reach a length of .10*mm*. The thickest are not usually the longest. None of the bodies contains definite nuclei, although occasionally, after alum hematoxylin staining, some resemblance to a nucleus is produced by a portion of the substance staining more deeply than the rest. These pseudonuclei never show nuclear membranes or contain chromatin nets or nucleoli.

The bodies stain intensely in eosin, acid fuchsin, or carbol-fuchsin, and under moderate magnification, they usually appear uniformly hyaline; but sometimes they consist of large hyaline balls. After Zenker's fixation, they stain intensely black in phosphotungstic acid hematoxylin, and when then seen under the oil immersion lens they always appear more or less granular. Frequently their bodies are continued into long processes, which in some instances are fairly coarse, but in others extremely delicate. Some of these processes stain like neuroglia fibrils throughout, others stain only along short stretches, or show granules along their borders.

In a few instances, a process after becoming unstained can

[1] In several diffuse gliomas of the brain, selected at random, I have found these bodies present, and in one tumor, abundant. General pathologists have evidently given them little attention.

be traced into connection with a neuroglia cell. As a rule, however, no such connection can be made out, and as the fibril is followed, it either appears to end abruptly, or to stain so feebly and to become so ill defined that its relation to a nucleus cannot be demonstrated.

In Case 1, in the thickened retina, near the tumor of the disk, where the neuroglia has a comparatively normal arrangement, the formation of cytoid bodies can be definitely traced. The earliest indication of one consists of a swollen portion of a cell process, filled with large eosinophilic granules (Fig. 12). This process is connected chiefly with one cell lying distant from it, but has communications with other cells also. In other words, cytoid bodies are formed within the trabeculæ of a neuroglia syncytium. In the early stages, the eosinophilic material does not stain so intensely as it does when it has become more abundant, so that it probably undergoes further chemical changes as it becomes older.

As these bodies stain differentially by Mallory's neuroglia method, and are frequently in continuity with fibrils indistinguishable from neuroglia fibrils, they can, perhaps, be looked on as atypical or giant neuroglia fibrils. Careful study shows that the stainable substance is deposited first in the form of irregular granules just beneath the cell membrane of the process. These increase in number and size until they completely fill the processes for a certain distance. The large cytoid bodies evidently result from distention of the cell processes by excessive accumulation of the stainable substance. For if enlargement of the process occurred first and the deposit of stainable material later, it should be possible to find such unstained swollen process in various stages of development. As a matter of fact, no swollen processes incompletely filled with stainable material can be found.

CLINICAL FEATURES

The clinical features associated with gliomas of the optic nerve have been fully described by Hudson. He found the tumors were more frequent in the female sex in the ratio of about 7 to 4, and that they occurred within the first decade of life in about 75 per cent. of the cases. In one case, the tumor

occurred as late as the sixtieth year. Hudson considered injury of considerable importance as a predisposing cause; but to me his evidence on this point is not convincing. Exophthalmos was of very slow development, the eye, as a rule, being protruded painlessly in the direction of the orbital axis. Limitation of ocular motion was generally not marked, but occasionally was complete. The optic disk usually showed evidences of papillœdema or optic atrophy. Vision was usually affected before the onset of exophthalmos, and was almost always completely abolished at the time of operation; but in one case it was said to have been normal, and in two others very slightly affected. Hudson gives no data regarding the frequency of occurrence of these tumors; but that they are extremely rare is indicated by the fact that only three such tumors have been removed at this institution within twenty-seven years.

In no case, except my Case 1, has the tumor certainly involved the globe itself. In the cases collected by Hudson, which came to necropsy, fifteen in number, more or less intracranial involvement was found in all, indicating that this is to be expected in at least the majority of cases. As Hudson points out, "The large size attained by the cerebral new formations without the production of severe symptoms has in several cases been very remarkable and is to be attributed probably to the very slow rate of increase." No case has yet been recorded in which the tumor has been confined to the intracranial portion of the optic nerve. Should such tumors occur, possibly they may show characteristics somewhat different from those of the tumors of the intraorbital portion. In many, if not the majority, of cases, the removal of the orbital tumor has been incomplete, yet recurrence in the orbit has never been recorded, although cases have been followed from two to twenty-four years.

As regards operation, Hudson advises removal of the tumor without the globe, if possible, through the soft tissue, except perhaps when there is still some vision and the diagnosis is doubtful, in which case Krönlein's operation is probably to be preferred. Complete removal is often impossible, owing to the frequency of intracranial involvement. Death from meningitis has resulted from operation in about 10 per cent. of the cases.

Considerable interest has been aroused by the marked pigmentary changes in the fundus, which generally follow removal of a tumor of the optic nerve without the globe. I have examined microscopically an eye in which the optic nerve had been cut several years previously in order to relieve the annoying diplopia resulting from a strabismus operation. In this case, there was extreme atrophy of both the choroid and retina, with pigmentation of the latter by cells migrating from the pigment epithelium. Since obstruction of the central vessels alone does not lead to such changes, it is evident that they resulted in this case from section of the posterior ciliary arteries and that the same explanation undoubtedly applies to the cases under discussion.

The occurrence of retinitis proliferans, after removal of the tumor without the globe, as in Case 5, is distinctly unusual, surprisingly so, in fact, in view of the marked interference with the retinal circulation which must result from such operations.

CONCLUSIONS

1. The most common tumors of the optic nerve are gliomas. These are the only primary intraneural tumors of this nerve that have been observed.

2. These tumors are composed of three main types of neuroglia which may grade into one another. Some tumors consist of all three types; rarely does one consist of a single type. Tumors in which so-called spindle cells predominate contain the largest and most conspicuous neuroglia fibrils.

3. The spongy structure often displayed by these tumors, which at times has led to a diagnosis of myxoma, myxoglioma, or myxosarcoma, is not the result of myxomatous degeneration, but is produced by excessive vacuolization of a neuroglia syncytium. Cysts of various sizes, which also often occur in these tumors, are due to the same process.

4. The tumor is probably essentially congenital in origin and dependent upon some abnormality in the embryonic development of the neuroglia of the optic nerve. The growth stimulates proliferation in all contiguous neuroglia tissue and thus causes the latter to take on the character of tumor tissue. In one case, the tumor in this way caused marked gliomatous

involvement of the optic disk and of the retina surrounding the latter. In another case, a glioma of the optic nerve was associated with a glioma of the *pars nervosa* of the pituitary body.

5. The theory that tumors of the optic nerve stem are related in origin to neurofibromas of the peripheral nerves is, at present, founded on insufficient evidence, but cannot be dismissed as impossible.

SYMPATHETIC OPHTHALMIA: REPORT OF TWO CASES CURED.[1]

By Dr. DERRICK T. VAIL, Cincinnati, Ohio.

SYMPATHETIC ophthalmia is insidious and silent in its onset, its progress is steadily onward, the results of treatment are disappointing in the vast majority of cases and the prognosis from its start is bad, so that cured cases when encountered should be tabulated and reported.

The literature on this disease where, in spite of excellent treatment blindness has supervened, makes gloomy reading.

To encounter two cases within six months' time in each of which a complete cure was accomplished is indeed a rare experience. In reading the notes of such cases we naturally wonder if the disease was genuine, and are inclined to feel that it may have been ocular syphilis, or uveitis or toxemic plastic iritis and not the true *Sympathetic Ophthalmitis*.

It would be well if we would formulate and agree upon certain definite clinical findings which must invariably be present in order to establish a correct diagnosis.

A positive diagnosis can be made in the laboratory from microscopic examination of the enucleated specimen but it is more important to be able to definitely diagnose the disease *in vivo* from early clinical manifestations.

It seems that the following clinical triad of objective findings must exist in order to establish an undoubted diagnosis:

I. A penetrating injury of one eyeball[2] followed by a lack

[1] Read before the first meeting of the Cincinnati Ophthalmological Club, Cincinnati, Ohio, December 9, 1921.

[2] An incision for the performance of cataract extraction or iridectomy fulfills this requirement as well as an accidental perforating injury.

of healing response, which is manifested by a quiet iritis and a disposition to wandering or detaching pigment cells from the uveal pigment of the iris, along with the formation of profound posterior synechia and absence of severe pain in the injured eye.

II. The presence of systemic anemia.[1]

III. The appearance in the fellow eye of the following:[2]

(a) Quiet iritis with rapidly forming circular synechia,

(b) Plastic optic neuritis and retinitis, and

(c) Minus tension as demonstrated by the tonometer.

The following two cases presented all these phenomena and I feel certain the diagnosis in each was correct.

CASE I.—On December 14, 1920, S. B., white, male, age thirty-three years, farmer, enjoying robust health, while at work on his farm cutting nails to detach boards from fence posts and while using a hatchet as a chisel held in his left hand and a hammer in his right, felt something hit him in the left eye. There was instant pain followed by blindness.

Examination seven hours after the accident:

There was a horse-shoe-shaped oblique perforating wound present in the left cornea opposite the upper surface of the iris. The iris behind this was torn, the lens was cataractous.

Operation: The wound of entrance was slightly enlarged with scissors. The giant magnet was applied and a large fragment of iron was drawn from the depths of the vitreous chamber and extracted through the wound. The torn and herniated iris was carefully abscised and the wound neatly dressed after a thorough cleansing with $\frac{1}{4000}$ solution bichloride mercury. The patient was put to bed at once and wet compresses were applied for the balance of the night.

The eye did not do well. There was, of course, marked inflammatory reaction. Wound infection and plastic iritis developed within three days, followed by glaucoma after three more days. As there was from the first no light perception present in the injured eye the patient was advised to have it enucleated. This he stoutly refused to permit.

Iridectomy near the wound was performed on the seventh day after the injury to relieve tension and the galvano cautery was employed at the same time to the lips of the infected

[1] This term is used to express a systemic impression that is invariably present in this disease, an impression characterized by palor and adynamia.

[2] These sympathizing symptoms rarely appear in the second eye before the seventh and usually after the tenth day following the traumatism.

wound. These measures did not bring about an improved state of affairs and the patient was again urged to submit to enucleation as a safeguard against sympathetic ophthalmia, but he again refused stating he would return home to spend the Christmas holidays with his family and return later on. At this time his right eye was normal to all tests.

He returned to see me after an interval of two weeks. The injured eye was entirely blind, soft, wrinkled, and sunken, the cornea was small, the iris was yellowish, and the anterior chamber was very deep. He made regular office visits after intervals of two weeks each, presenting the same unimproved appearance as regards the injured eye. At each of these visits the right eye was examined and found to be normal to all tests.

On March 29, 1921, over three months after the accident, I took a firm stand and told him it was positively dangerous to delay enucleation so long but he refused stating there was nothing wrong with his good eye and that he desired for esthetic reasons to retain his injured eyeball even if it was unsightly and dangerous.

I then told him if he noticed transitory attacks of shadows or "showers of soot" with his right eye he should come to see me at once.

On April 11, 1921, two weeks after this or four and one-half months after the accident he came in an excited state of mind because he had experienced two attacks of momentary blindness of his right eye the day before and it frightened him to be blind like that even for a moment. He determined to have the injured eye removed at once, for on his way to Cincinnati he had had two more similar attacks, so that he could hardly wait but wanted the enucleation operation done at once.

I found the group of objective symptoms in the second eye mentioned in the beginning of this report—ring synechia was already established, neuro-retinitis was positively present. The dioptric media were slightly misty in appearance, the tension was 18*mm*, the vision was $\frac{20}{30}$, the fields of vision for white and colors were normal. There was no pain present but slight deep pericorneal injection was noticed with focal illumination aided by the binocular loupe.

I enucleated the first eye at once and ordered a brisk calomel purge followed by twenty-grain doses salicylate of sodium every four hours. The patient was kept in bed. A two per cent. solution atropine or a one per cent. solution of scopolamine was instilled alternately in the remaining eye every four hours for two days. The synechiae were thus all separated and the pupil became fully dilated and circular. The fundus likewise appeared improved and the

patient was permitted to return home after a week and was advised to repeat the calomel or castor oil purge twice a week together with the twenty-grain doses of sodium salicylate every four hours for three days out of every week.

He returned eleven days later with the remaining eye presenting a nearly-normal appearance, but on May 10, 1921, thirty days after enucleation, he returned again presenting the typical ocular picture of sympathetic ophthalmia. No symptom was lacking. The synechiae had reformed, the neuro-retinitis was again in evidence, the deep ciliary injection returned, the eyeball was soft (tension 16*mm*) and things looked bad indeed.

A Wassermann test was made and found to be negative. It was at this stage that I noted the extreme facial pallor of the patient. When he first came he was robust and rubicund; he now presented the striking picture of anemia and asthenia.

The patient was put to bed, atropine and scopolamine drops were used as previously, active eliminations with castor oil followed by epsom salts employed, and sodium salicylate in twenty-grain doses again used every four hours. In four days' time the pupil was again dilated fully excepting one synechia below which resisted all efforts to sever.

I consulted Dr. Faller of Cincinnati, an expert hematologist, to learn if salvarsan would have a beneficial effect in combating anemia of the kind the patient exhibited and if so whether it was safe to employ it in non-syphilitic patients. I had heard it had been used with benefit in such cases. He assured me that because of the arsenic content it would have a positive effect in improving and building up the red cells of the blood and that it would be safe to use it in half-sized doses. (0.15 gram.)

He was given this fractional dose of salvarsan intravenously. The effect was striking. In two weeks' time he returned with good color in his face and his eye was clear and bright, vision being normal. The salvarsan dose was repeated and in another two weeks the cure became permanently established. The patient took twenty-grain doses salicylate of sodium three times a day for three days out of each week for a month's time after the second and last dose of 606, and then, as he seemed well, he stopped it of his own accord.

I have seen this patient at monthly intervals for eighteen months after all medication was ceased and his eye remains entirely normal to all tests. The tension rose to 22 and 24*mm* and remains normal.

COMMENT: The above case was undoubtedly genuine, but it is difficult to determine exactly what brought about the com-

plete cure. Enucleation of the first eye within forty-eight hours after the disease announced itself in the second eye unquestionably removed the "storm center" but clinical experience has proven that enucleation alone has little effect upon curing the disease which has already started in the fellow eye. No doubt the salvarsan should have the credit of overcoming the anemia, and the elimination treatment employed, together with the medication with sodium salicylate, may have combated the toxic agent present in the blood.

CASE 2.—Mrs. O. H. F., housewife, age forty-seven, in rather delicate health on account of what seemed to be chronic toxemia or bacteremia associated with striking pallor of the skin of the face or, as called by former clinicians, "cachexia," came to see me on March 31, 1921, on account of an attack of fulminating glaucoma with much pain, redness of the eyeball and nearly total blindness of her left eye of less than twenty-four hours' duration. The tension of the left eye was 70*mm* as measured with tonometer. There was the typical picture of violent acute inflammatory glaucoma, *e.g.*, steamy cornea, obliterated anterior chamber, fully dilated and fixed pupil, greatly impaired vision, chemosis and redness of the eyeball together with the typical severe pain affecting the upper branches of the fifth nerve.

There was a family history of glaucoma, for the patient's father had become blind from this disease, her uncle had also lost his vision presumably from chronic non-inflammatory glaucoma, and another uncle had gone blind from cataract "with complications," so that she was mentally prepared for the worst and seemed to be resigned to any fate.

The right eye was normal to all tests, vision $\frac{20}{20}$ without lenses.

Operation: Iridectomy, under local (subconjunctival) anæsthesia was successfully performed within twenty-four hours. As the result of this operation the tension fell to less than normal but an astonishing phenomenon resulted. The lens which from the high tension had been crowded against the posterior surface of the cornea, to which it seemed to be adherent, refused to return to its bed. It was dislocated in the anterior chamber. The iris fell back so that the *lower part receded behind the lower edge of the lens*. The pupil was fully dilated but the tension remained normal. In nine days' time the lens began to swell from acute cataract formation and I was compelled to advise its removal.

Operation on April 16, 1921: Sixteen days after the first

operation the cataract was successfully extracted. The anterior capsule remained adherent to the posterior surface of the cornea at its upper part and so the capsule was retained within the globe being dislocated upward, however, so that there was a perfectly clear space below it in the axis of vision. With suitable lenses the vision was $\frac{20}{20}$. The field of vision was normal; and fundus likewise appeared normal. It looked like a most happy result after all, but on May 27th, which was six weeks after the cataract operation, glaucoma suddenly returned and the tension rose inside of twenty-four hours from *22mm* to *46mm* mercury. I advised the iridectomy operation at once.

Operation, May 28th: Iridectomy of both arms of the coloboma was successfully performed under local anæsthesia employed subconjunctivally and the tension was by it reduced to below normal again.

The right eye was inspected at each dressing for I was always fearful of glaucoma appearing in it. The family history as to glaucoma was bad and the disease had appeared spontaneously in her left eye and without known cause.

On the seventh day after the last operation (viz. on June 4th) I noticed slight ovality of the right pupil. The eyeball was perfectly pale, the vision was normal, but the pupil was contracted and as stated was of oval contour. Examination by focal illumination aided by loupe revealed well established ring synechia. The tension by the finger test seemed soft, not over *18mm* (estimated). I suspected sympathetic ophthalmia at once and called for a consultation with Dr. Clarence King.

Dr. King agreed that a quiet iritis was established in her second eye. We desired to use a mydriatic but how could we safely do so with the history of glaucoma so recently in the case? Finally Dr. King and I agreed to use euphthalmine or homatropine cautiously.

Seven per cent. solution euphthalmine dropped in the eye dilated the pupil slightly, demonstrating a crenated pupil margin. Two per cent. solution of homatropine drops dilated it still more, demonstrating at first a daisy-shaped pupil, finally a kidney-shaped pupil, and thus one by one all the synechiæ were separated within twenty-four hours excepting one strong one below which resisted.

Examination of the fundus was now possible and I discovered a positive plastic optic neuritis and peri-neural retinitis. One exudation was very striking near the disk margin at about "one o'clock." The macula lutea was distinctly œdematous, fine precipitates were observed in the aqueous chamber. The vitreous presented fine opacities in

great numbers. The tension with McLean's tonometer was 19*mm* mercury.

Dr. King thought toxemia or chronic septicemia was a possible cause and that perhaps the trouble was more in the nature of a toxemic neuro-retinitis and iritis.

Dr. J. E. Greiwe, a prominent internist, was summoned to examine and report on the patient's general physical state. Dr. Greiwe made a thorough physical examination including blood count and urinalysis, reporting his findings in writing. He conducted a Wassermann test and found it to be negative. He discovered abscesses at the apices of six of her teeth by X-ray examination and finally returned a diagnosis of secondary anemia probably due to chronic bacteremia—recommendng intravenous injections of the citrate of iron (0.10 in 1*cc* hypule form).

At this juncture Dr. Harold Gifford of Omaha was called in consultation.

Dr. Gifford made a thorough examination of the patient's eyes and finally diagnosed sympathetic ophthalmia. He agreed that the citrate of iron should be given and repeated every second day until the anemia was overcome. He strongly urged that the patient, whose body weight was less than one hundred and twenty pounds, be given one hundred and twenty grains salicylate of sodium every day without ceasing for weeks and months if need be or until convalescence was established. He advised atophan as a good substitute to be given *per os* in case the sodium salicylate disturbed the digestion.

He considered the advisability of removing the first eye. It possessed $\frac{20}{200}$ vision at that time and a normal peripheral field of vision by the candle test, and so he advised against enucleation, stating that if blindness of her second eye supervened the first eye might be the better of the two.

It would make tiresome reading to publish all the notes from this on. Suffice it to say we secured written instructions from Dr. Gifford and gave the patient the dosage he advised.

The patient took sixty grains of sodium salicylate every morning *per rectum* and thirty grains of atophan *per os* every day after the noon meal; in addition to this she took an extra dose of sixty grains of salicylate of sodium *per rectum* at bedtime every third day. This was the program she followed for three months, ***ninety days without a break***, and after that the same doses were taken every second day for thirty more days and then twice a week for another thirty days.

For two and one half months she was given every second day the 0.10 citrate iron intravenously by Dr. Greiwe.

Ten days after Drs. Gifford and Greiwe's treatments were

instituted there were signs of a clearing-up process in her second eye. The fundus looked brighter. Her vision which had fallen to $\frac{2}{40}$ became $\frac{3}{20}$.

On July 21st, or six weeks after consultation with Dr. Gifford, a remarkable phenomenon occurred. The firm synechia melted away and disappeared leaving the pupil perfectly round and mobile throughout. This phenomenon took place without the aid of a mydriatic. Moreover *every vestige of ring deposit of pigment and lymph on the anterior capsule of the lens disappeared. The precipitates in the aqueous and the opacities in the vitreous humors all disappeared. The fundus cleared up as regards all the exudations and cloudy swellings.* The tension became normal (24*mm* mercury). The vision became normal, $\frac{3}{20}$ + and Ja. 1.

Within two months after the treatment of Gifford and Greiwe was instituted the second eye seemed to be cured.

On November 16, 1921, seven and one half months after the disease began, all treatment was withdrawn and the patient pronounced cured as regards her second eye.

I had repeatedly suggested the employment of salvarsan in this case to combat the anemia but it was refused by her husband who, knowing what the remedy was used for, would not allow it to be employed.

The six offending teeth were drawn in three seances of two weeks' interval for it was feared that to draw these all at once might have a tendency to liberate septic poisons in the blood in damaging quantities.

The *first eye* has a normal field of vision by candle test but no quantitative vision is present on account of *occlusio pupillæ*. The tension is normal, eyeball is pale like the second eye, and the cornea is clear, but the iris is bombé. The space of the coloboma is filled with gray organized exudate completely blocking the pupil.

It is too soon to state that no further trouble will develop in the first eye; most likely it will become glaucomic, but the second eye is apparently entirely cured and should remain so.

If the first eye should develop glaucoma again we all agree that the only operation to consider for it will be enucleation.

COMMENT: This is a remarkable case presenting all the clinical manifestations of genuine sympathetic ophthalmia. The first eye was not removed and hence must have discharged large quantities of protein poisons in the blood, all of which were overpowered by the persistent use of large doses of salicylate of sodium and its substitute, atophan, aided no doubt by the upbuilding of the red blood cells by the action of citrate

of iron thrown directly into the blood stream by intravenous injections. Her anemia was overcome. She gained in weight and lost the excessive pallor of her skin. The removal of the teeth that presented apical abscesses may have removed the source of provocative or contributory toxemia. In all events the cure is complete.

THE USE OF THE GULLSTRAND SLIT LAMP.[1]

By Dr. HARRY S. GRADLE, Chicago, Ill.

(With one illustration on Text-Plate XIV. and seven in the text.)

THE value of the slit lamp depends upon one of the simplest principles known to physics, so simple that it was overlooked until Gullstrand adapted it in the instrument that has added so much to our knowledge of the human eye. The principle is that of simultaneous contrast. A particle of dust floating in the air of a diffusely illuminated room cannot be perceived because of the lack of contrast. But allow a beam of sunshine or any concentrated light of sufficient intensity to pass through the room and the particle of dust becomes visible immediately and clearly, due to simultaneous contrast. The same principle holds true in examination of the tissues of the living eye with varying degrees of magnification. A diffuse illumination does not allow the particle of tissue in question to stand out in contrast to neighboring particles. But the concentration of a beam of intensive light upon an area, at the cost of illumination of adjacent areas, reveals details of structure hitherto unknown. Such is the power of focal illumination and simultaneous contrast.

Microscopy of the living eye is accomplished by means of a combination: a microscope, and a focal illuminant.

The binocular microscope is not of recent origin, although many improvements have been added to increase the efficiency. In 1899, Czapski developed the microscope that to-day bears his name. It consists of two convergent tubes with a double objective, and two oculars. The rays of light from a point

[1] Read before the American Acad. Ophth. Oto-Larygn., Philadelphia, Pa., October 20, 1921.

ILLUSTRATING DR. HARRY S. GRADLE'S ARTICLE ON "THE USE OF THE GULLSTRAND SLIT LAMP."

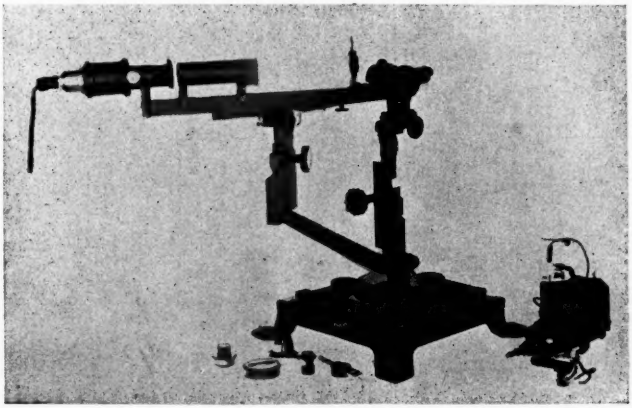

FIG. 1.

pass through the converging objectives and fall upon a pair of Porro prisms, which reverse the image and bring it into an upright position and at the same time allow the tubes to be swung apart or together to coincide with the interpupillary distance of the observer.

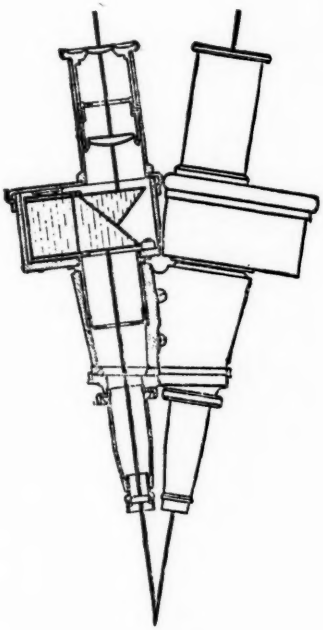

FIG. 2.

Schematic cross section of binocular corneal microscope.

The rays are then continued through the well-known Huygens oculars, which collect the main rays diverging from the objectives, which enlarge the image received from the objectives and bring the rays to a parallel or converging course, in order that they may be properly focused upon the retina of the observer. By changing the objectives as well as the oculars, magnifications ranging from twenty to one hundred and three diameters can be obtained.

For examination of the deeper recesses of the eye, a modification of the corneal microscope, known as the "Bitumi," has been devised. This consists of a single objective of somewhat greater length, a single microscope tube, a set of half silvered glass plates, which function essentially the same as the glass prisms in the Abbe stereoscopic ocular, and a pair of parallel oculars. The interpupillary distance of the observer is accounted for by a simple device, which separates the parallel ocular tubes the required distance.

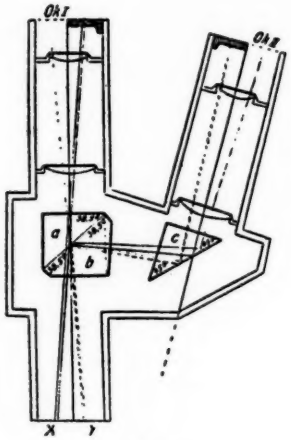

FIG. 3.

Schematic course of rays through Abbe stereoscopic ocular.

The Bitumi presents the following advantages over the binocular corneal microscope: (1) greater magnification, even beyond the limits imposed by the involuntary movements of the eye under observation, and (2) penetration into the deeper structures, that are without the pale of the Czapski, because of the separation of the entrance pupils of the converging tubes. On the other hand, the Bitumi is somewhat more difficult to handle (the image is reversed) and there is a considerable loss of light.

The illuminant is a 25-volt Nitral lamp, operating upon a 110-volt current with an interposed resistance. The lamp

itself has a tightly wound spiral filament within a rather small gas-filled chamber, and presents as nearly as possible a vertical line of light. The divergent rays are rendered parallel by a single planoconvex lens, and are again converged by a

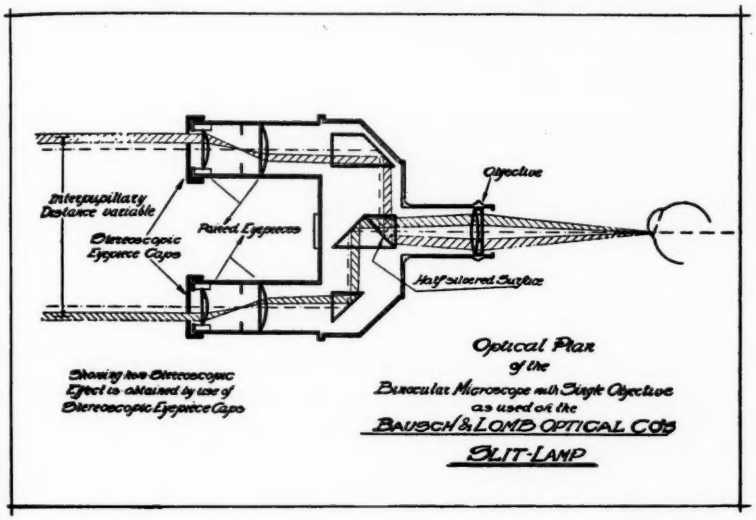
Objective
Interpupillary Distance variable
Paired Eyepieces
Stereoscopic Eyepiece Caps
Half silvered Surface
Showing how Stereoscopic Effect is obtained by use of Stereoscopic Eyepiece Caps
Optical Plan of the Binocular Microscope with Slight Objective as used on the
BAUSCH & LOMB OPTICAL CO'S
SLIT-LAMP

Fig. 4.

planoconvex lens upon a slit of variable dimensions. The spherical aberration is to a great extent eliminated by having the spherical surfaces of the lenses facing each other. Thus an image of the luminous filament is formed in the slit.

The almost parallel rays of this image are then brought to a linear point by the condensing lens at the end of the lamp arm. Lateral diffusion is eliminated by the tube interposed between the slit and the condensing lens, which tube also serves to carry various filters that are used in special examinations. For penetration into the deeper structures of the eye, a metallic mirror is set in an upright at the extreme end of the lamp arm, beyond the condensing lens. When it is necessary to make the angle of convergence of the axis of illumination and the axis of observation as small as possible, the lamp arm is put at right angles to the axis of observation, and the

illuminating beam is turned at right angles by the mirror. This eliminates mechanical difficulties.

Exact centration of the lamp is essential. The lamp socket must be rotated until the filament is exactly vertical and then locked in place by the fixation screw. The slit must also be vertical and so placed that the image of the slit falls in the

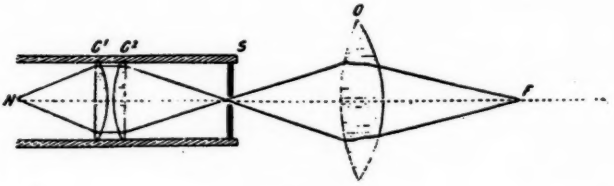

FIG. 5.

Schematic course of rays in the illuminating apparatus.

exact center of the condensing lens. Lateral movement of the slit is provided for by a helical arrangement. In the beginning, it is preferable to open the slit as wide as possible, but for varying conditions the width may be reduced to advantage.

The patient must be seated comfortably with the chin resting firmly upon the chin rest, and the head braced against the forehead rest. Strained and unnatural positions should be avoided. The patient should be instructed where to look, and in many cases the small fixation lamp attached to the instrument is of great value, particularly in examination of the fundus. To obtain the first rough position of the microscope, the eye may be sufficiently illuminated by withdrawing the lamp until the broadened slit covers a fair share of the cornea. The microscope should then be moved by means of the double adjustable base until the primary rough focus is obtained. The lamp is then moved forward until the image of the slit appears upon the cornea, in approximately the focal size. The final fine adjustment is made with the rack and pinion that operates the condensing lens upon the lamp arm. The observer, who by this time has adjusted the microscope to his own interpupillary distance, focuses the microscope upon the tissue under examination. If the left eye of the patient is being observed, the lamp is upon the observer's right, and

his right hand is occupied with the rack and pinion adjustment of the condensing lens and with the lateral motion of the base, while his left hand manipulates the fine adjustment of the microscope and keeps it trained upon the area in question. The reverse position obtains for the other eye.

Four types of illumination come into consideration. The first is the basic principle of the instrument, ***direct focal illumination***, and has already been discussed. The second is illumination by *indirect focal light*. During the observation of any specific area by direct focal illumination, it will be noticed that contiguous areas can be seen and in a more plastic form, owing to their illumination by reflected and diffused light from the more brightly illuminated zone. This method is particularly of value for the study of delicate opacities. The third is ***dark field illumination***, and applies of course only to the transparent or translucent areas of the eye. The focal point of light is directed upon an area behind the structure to be observed, which then stands out in the well-known dark field form. The examination of structures with different indices of refraction, as blood vessels or nerves in the cornea, is facilitated by this method. The fourth, and but little used, type is observation in an ***oscillating light field***, where the oscillations are produced by manipulation of the lamp arm.

For examination of the background of the living eye, a slightly different technic is employed. The lamp arm is placed at right angles to the axis of the Bitumi, and the beam of light is bent at slightly more than ninety degrees by the metallic mirror at the end of the lamp arm. The illuminating bundle thus forms a sharply convergent bundle, with the axis of observation and the mirror in juxtaposition with the tip of the microscopic objective.

In order to bring the image of the background forward to within the range of the microscope, it is necessary to use a contact glass of the type designed by Fick. Such a glass fits directly over the cornea, and has a posterior radius of curvature of 8*mm* and a diameter of 12*mm*. The anterior surface of the glass is ground flatter than the posterior, has a radius of 10*mm* and an equivalent value of +5D. This makes the refraction of the glass equal to − 59.5D., and brings the image of the retina some 16.7*mm* behind the posterior surface of the

cornea, or about in the middle of the anterior third of the vitreous. This image is slightly smaller than the true retina (0.96).

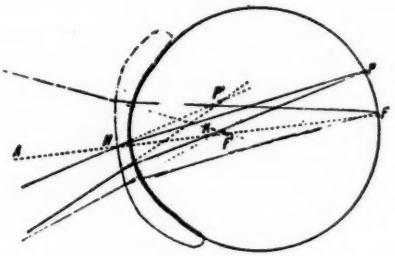

FIG. 6.

Schematic course of rays through contact glass, showing how the retina is brought forward.

For such observation, it is necessary to have the eye in good mydriasis and well anæsthetized. The patient's head should be tilted forward, and the lids opened with the fingers of one hand. The contact glass, previously filled with warm normal salt solution, is then applied directly over the corneal, which must be tilted slightly downward. Immediately upon application of the glass, the patient must look directly forward and the lids be allowed to close spontaneously. (It is almost unnecessary to add that fingers must not touch the observation surface of the glass, because of resultant diffusion by adherent grease.) If a bubble lies between the glass and the cornea, the procedure must be repeated, or else the upper edge of the glass can be lifted away from the sclera with an iris spatula, and a thin stream of salt solution allowed to fill in from a small pipette. The flattened portion of the glass should be directly over the pupillary area of the cornea, and should there be a tendency to drop, the glass can be held in place by light pressure on the lower lid. Removal of the glass is accomplished by lifting the upper edge with the spatula, whereupon the upper lid in closing forces the glass out. Care must be taken to avoid dropping or scratching the glass.

After the contact glass has been applied, the patient is seated before the Bitumi as previously described, and the lamp arm and metallic mirror adjusted so that the focal slit falls first upon the edge of the iris, and then 14 or more *mm* behind it. The fine adjustment must be made with the entire apparatus rather than with the condensing lens. The Bitumi is then moved into place, and the brilliantly illuminated fundus comes into view. The same adjustment holds for the examination of the vitreous, merely changing the focus of the different instruments.

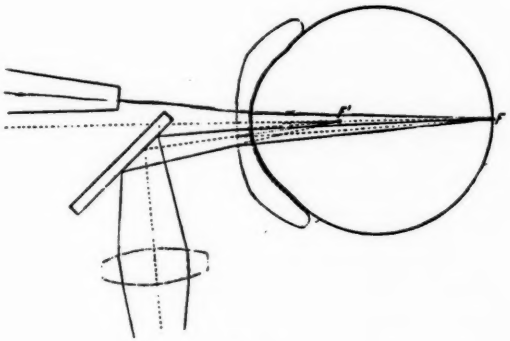

FIG. 7.

Schematic course of rays in microscopy of the human fundus.

The angle of the anterior chamber may be examined in the same manner, although the use of a miotic is necessary, and the patient's head must be turned about 45 degrees on the chin rest. A different type of contact glass is used, with a concave anterior surface ground upon a heavily convexed surface. The illumination enters through a very sharp angle, while the observation axis is nearly parallel to the surface of the iris. Needless to add, it is much simpler to examine the chamber angle from the temporal than from the nasal aspect.

From the foregoing rough description, it can be seen that many new phases of examination of the living eye are made possible by the use of the slit lamp of Gullstrand. For daily clinical use, the binocular microscope and the first three types

of illumination mentioned afford a much more intimate knowledge of pathologic processes of the anterior eye than was ever deemed possible, and practice renders such examinations fairly rapid. The use of higher magnifications, and of the apparatus

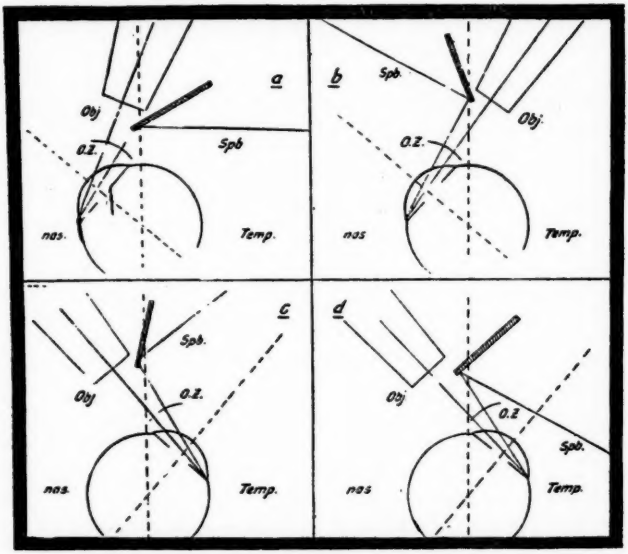

FIG. 8.

Schematic course of rays for chamber angle microscopy.

for examination of the chamber angle and of the background, is somewhat more laborious, but in many obscure cases the time thus spent is well recompensed by the more intimate knowledge gained.

Many of the schematic illustrations used are taken from Koeppe, *Die biophysikalischen Untersuchungsmethoden der normalen und pathologischen Histologie des lebenden Auges.*

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, LONDON.

A meeting of the Section was held on Friday, January 13, 1922, under the presidency of Dr. JAMES TAYLOR.

Sir John Herbert Parsons's Knighthood.

The PRESIDENT felicitated Sir John Herbert Parsons on the honor of Knighthood, which the King had recently conferred on him, and in the name of the Section expressed the hope that Sir John would live long to enjoy it.

Sir JOHN PARSONS, in returning thanks, said the honor was a recognition of his having served on a number of Government Committees, on which, however, he only served as an ophthalmologist, therefore it was an honor to ophthalmology, and he was the fortunate peg on which it was hung.

Cases.

Mr. DOYNE showed a case with a **congenital deformity of the conjunctiva,** associated with deformities of tongue and thumb in the same patient.

Mr. REA showed a case of **filamentary keratitis,** which lasted three years and had been very resistant to treatment. When bullæ were removed, the patient came with a fresh crop in three weeks. Tension was normal, and there was no suggestion of glaucoma. The woman had suffered from arthritis for many years.

Mr. HUMPHREY NEAME showed a case with branching remnants of persistent hyaloid vessels. He thought it likely

that these processes arose from one common trunk. Vision in the right eye, with correction, was $\frac{6}{36}$, in the left $\frac{6}{9}$. There was also a thickening of the walls of some of the arteries in the right fundus; the strands had no connection with the lens capsule.

Miss MANN exhibited slides and drawings of embryonic eyes showing the earliest formation of a definite hyaloid artery. Until just before five weeks, she said, the globe was filled with mesoderm in the meshes of which there were blood vessels but definite branching arteries could not be traced much below five weeks. There were usually five branches of the vessel, and they broke up again into blood spaces, which quite surrounded the lens. A 3 months' embryo showed a differentiation of retina, with optic nerve. From the center of it came a definite hyaloid artery with thick walls. That broke up into five branches. She next showed an equatorial section 6*mm* in front of the disk; it showed a division into three main branches. In the next section, 1*mm* in front of the last, there was again a division into five. That went on to the sixth month, after which it began to disappear again.

Mr. M. S. MAYOU referred to injected specimens of hyaloid artery in embryo which he did some years ago. At the 4th month it could be seen that the hyaloid did not break up at the back of the lens, as so many text-books depicted, but far back in the vitreous. The posterior vascular capsule was formed by a cone of vessels passing up to the back of the lens. In some cases one branch was found persisting and anastomosing with the ciliary body. Sometimes there were masses of fibrous tissue at the back of the lens, which were supposed to be remnants of the hyaloid artery.

Mr. LESLIE PATON suggested that Mr. NEAME's case might be one of proliferating retinitis.

Mr. NEAME replied that though Mr. Paton's suggestion was a likely one, the five branches seemed to come so locally from the back that this seemed to negative that idea.

Mr. M. S. MAYOU exhibited a baby with **acute necrosis in the upper jaw.** This was his third case of the kind, all under one year old. One had successive portions of the jaw removed until none remained; it was a spreading necrosis. It was an acute process in all the cases, and he believed the mouth could

be excluded as a channel of infection in the present case; moreover the disease occurred before teething, and before sinuses were present in the bone.

Mr. LESLIE PATON mentioned a case of his own of the same character, a healthy breast-fed child nearly four months old. When he first saw the child there was a large swelling under the left eye, and the eye was proptosed forwards and outwards to the extent of an inch. He evacuated about an ounce of pus from behind the eye. Bare bone was evident to the probe, but there had not been time for necrosis to have taken place. The condition seemed to belong to the same category as acute osteomyelitis in infants.

Mr. STACK enquired whether syphilis and tubercle could be excluded.

Mr. PATON replied that those diseases could be excluded in his own case; the only organism found in it was staphylococcus.

Mr. MAYOU, in his reply, said both parents in one of the cases proved negative to Wassermann, in another the doctor was so sure there was no suspicion of syphilis that the test was not applied. The cases were, he thought, too acute for tubercle to be the cause; one had a temperature of 103°, and a pulse of 130.

Mr. LANG showed an operating lamp; it could be attached to the ordinary bracket and carry a ½-watt lamp, with the necessary resistance. It enabled fluorescine staining, such as in corneal ulcer, to be well seen.

Trephine Operations for Glaucoma. Experience Gained in 140 Cases.

Mr. MALCOLM L. HEPBURN read a paper entitled **Experience Gained from 140 Trephine Operations for Glaucoma.**

He regarded glaucoma as such a serious condition, especially the chronic type, with its variety of symptoms and signs, that the question of operative interference merited all possible discussion. Recently contributions had appeared favoring iris-inclusion and silk-inclusion operations, somewhat to the detriment of the iris-free operations. The reporting of late infections had deterred some surgeons from confidently recom-

mending trephining, and had led some to give it up. In perforating wounds of the eye, and after cataract extractions, surgeons tried to avoid permanent inclusion of iris in the wound because of the danger alleged to be associated with such inclusion, and he could not see the justification of it in operations for glaucoma, unless other methods had failed. The danger of leaving an opening in the eye separated from the external air by only a thin layer of conjunctiva was admitted, but it applied to all filtering scars, and the only question was as to which was the least dangerous.

In the 140 trephine operations he had performed in the last nine years, 27 were in private patients; and if the combined hospital experience of his colleagues could be studied, he thought there would be overwhelming evidence in favor of the trephine operation. At Moorfields, at least, late infection was very rare, and he believed its occurrence in a general way had been much exaggerated. He had not been without complications in his cases.

With regard to the technique he followed, the importance of an efficient conjunctival flap was very great in striving to avoid late infection; he made the flap as thick as possible, and he did his best to prevent tearing or button-holing of the conjunctiva, stripping the conjunctiva off the globe in its whole thickness down to the sclera from the beginning, and continued cutting with the scissors, the points of which were kept directed towards the globe. Toothed forceps he used only for the initial fixation, subsequently those employed were very fine toothless ones. On reaching the limbus he used the secondary cataract knife, keeping it directed towards the globe and pressing slightly. He never found that he got too far forwards, as he always found himself farther back at the end of the operation than he expected to be. Before commencing rotatory movements he drew the flap upwards and backwards so that it was parallel to the trephine, so avoiding button-holing. While rotating the trephine, he directed the handle forwards so as to cut through the anterior part of the scleral disk before the posterior to ensure the formation of a hinge posteriorly when the section was complete; then the disk could be removed later without fear of injuring the conjunctival flap. Having once got the trephine to bite, he did not remove it until

the section was finished, so that the full force of the aqueous discharge would push the knuckle of iris well out of the wound. The guide to the penetration of the sclera was the coming up of the pupil towards the hole. When the iris presented, it must be dealt with at once; the disk could be left to take care of itself. He grasped the iris with very fine straight forceps, at the same time pulling downwards and forwards, so as to detach it from its root. Lastly, he cut off the disk, which could be done easily without endangering the conjunctiva. Usually he inserted one or two stitches in the conjunctival flap; perhaps there should be more.

Proceeding to the consideration of complications, Mr. Hepburn said that if a hole was seen in the flap, a new point of application of the trephine must be selected. Three or four times loss of the disk had happened to him; it might be drawn into the anterior chamber, it might be left in the trephine, or it might be washed away when the aqueous was discharged. Sometimes the iris was so dilated at the time of operation, that the force of the aqueous discharge caused a total prolapse to the pupillary border; in this case all the prolapsed part must be cut off and a complete iridectomy performed. On occasion a complete iridectomy occurred from a too free division. If a complete iridectomy was intentionally done, the iris must be pulled well out of the wound. Apart from cases of buphthalmos—in which the accident was not uncommon—he had had vitreous loss only three or four times. Whether it influenced the subsequent draining capacity depended somewhat on whether the vitreous was solid or liquid. In two of his cases there was good drainage and no affection of vision afterwards; in one case the vision had remained at $\frac{6}{6}$.

With regard to delay in the re-formation of the anterior chamber this had occurred in about six of his series of cases. He now allowed these patients to be out of bed at the usual time after the operation, when he usually found that the anterior chamber formed immediately. When this formation was delayed unduly, he was anxious lest there might be adhesion of the iris to the trephine hole; for this reason he always used atropin as a routine treatment. In two or three of his cases the lens had come forward, but only in one case was he able to assure himself that there were no opacities previously. He

was not aware of detachment of choroid having happened often in his cases, but it might occur without being recognized. He had not encountered serious complications from this cause, though it might account for failure in the re-formation of the anterior chamber.

What cases were to be included in the term "late infection"? As the permanent opening in the eyeball was the weak point in the operation, the entry or organisms through this channel must be established to justify the use of the phrase; he thought inflammatory reaction must be associated with rupture of the conjunctival flap. Inflammations occurring many years after operation when there was a thick and firmly attached flap could not be included as late infections. And he did not see why every type of inflammation occurring in an eye after trephining should be directly ascribed to the operation.

A general review of his cases showed that by far the best results ensued in cases which he secured early, and especially if he had been able to do the operation at a time when the tension of the eye was normal. In acute glaucoma he only trephined if he had been able to reduce the tension before operating. If he could not reduce it, his practice now was to perform iridectomy. He had seen two eyes in the same patient in which one had been trephined in an early stage, but in which operation on the other eye had to be postponed, and the unoperated eye steadily went downhill with the same symptoms; but after operation these symptoms were cut short and controlled. That his private cases had been more successful than the hospital ones he attributed to the fact that private patients were more observant and anxious about initial symptoms, and early sought advice. His 13 failures in the 140 had been in cases whose tension at the time of operation was raised, when there was a long history of the trouble, and in which the field had been contracted in a general way, almost to the fixation-point. As failures he reckoned those cases in which sight was restricted to finger-counting, with much contraction of the visual field, and where no improvement followed the operation. Many cases appeared to have worse vision immediately after the operation but it improved later. He seldom had good results in acute or in secondary glaucoma; both these kinds showed too much congestion round the limbus

and this encouraged healing of the trephine hole. Moreover, in acute glaucoma the conjunctiva was often very friable. He had had two cases of cyclitis; both recovered. He had had no case of sympathetic trouble following trephining.

As a result of his experience, he had formed the opinion that the operation of trephining, performed with every consideration for the conjunctival flap, was the ideal one for chronic glaucoma, especially for the type generally met with in young adults.

Mr. R. R. CRUISE said all surgeons naturally preferred the type of operation which, in their hands, had been successful. He had now ceased to do trephining for glaucoma, though at one stage in his practice he trephined all his cases of that nature. He abandoned the operation because of one or two disasters, and since he had changed his operation those had not happened. In two there was an escape of vitreous; not, he thought, through any fault in technique. At the operation on one of them a colleague was present and praised the operation, foretelling a good result. He called in colleagues in consultation. The eye remained quiet, but vitreous continued to come out. He cautiously sealed up the hole, and the tension rose to *plus* 3 or 4, and the lens was drawn up. It was the patient's only eye, and he went blind. In two cases there was infection afterwards, causing conjunctivitis; these patients were left with vitreous opacities. He had also had, when doing trephining, a good deal of subconjunctival thickening round the trephine hole margins. For ten years he had been doing a modified flap sclerotomy, doing rather more than Herbert did, *i.e.*, completing the two sides of the flap well forward into the cornea, and beyond the rim of resistance felt when cutting through. It was very important not to suture the conjunctiva. One of the features of trephining which made him more dubious about it was the transparent pedicle which remained, enabling one to look into the depths of the eye.

Mr. M. S. MAYOU agreed as to the transparent bubble which was left after trephining, and it was a spot very difficult to cover and protect. A further objection was that the cornea had to be split in order to get the trephine on properly. During the last three months the speaker had been carrying out the

new operation of Holt, who claimed it did not leave a transparent bubble and that was true in the cases in which Mr. Mayou had so far done it. He described Professor Harden's method of ascertaining detachment of the choroid. The examination, done as early as the second or third day, was carried out by means of the contact-illuminator. This was put on the globe 5 or 6*mm* behind the sclera, and if there was a detachment of choroid, the whole area of sclera right to the cornea would be illuminated; but if the choroid was in its place there would be illuminated only an area around the point of contact.

Mr. LANG thought the danger arising from the hole in the flap had been much exaggerated. The amount of drainage was directly proportional to the diameter of the trephine hole. The operation he did for glaucoma was a modified Lagrange, turning back a piece of conjunctival flap and taking away a part of the cornea.

Mr. HEPBURN, in the course of his reply, considered that Mr. Cruise had had bad luck in his cases which caused him to adopt a different procedure. He, the speaker, did not find transparent blebs were common, and when they did occur they got thicker with the passage of time. He had never found that splitting of the cornea made any difference. The Lagrange operation was, in his opinion, merely a glorified trephine operation. Though patients who had a hole in the conjunctiva might survive trouble or escape it, it was not wise to design a procedure on that assumption.

Epibulbar Sarcoma, with Penetration of the Globe.

Mr. HUMPHREY NEAME read a paper on this subject, illustrated by a number of micro-photographs, projected by the epidiascope. In May, 1919, the patient noticed a swelling under the upper lid of the left eye. There was a history of syphilis, of recent ulceration of the palate, and deformity of the nasal bones, and the Wassermann reaction was positive. For some time the swelling was regarded as a gummatous infiltration but as it did not react to anti-syphilitic treatment, and the eye was practically blind, Sir John Herbert Parsons, under whose care the patient was, decided to enucleate the eye. Patho-

logical examination revealed an extensive epibulbar growth, with extension within the globe. The growth surrounded the cornea and, as a thin sheet, spread around the eye to the posterior pole. The iris, ciliary body, and choroid were completely infiltrated with the same type of growth. It was a round-celled sarcoma, with slightly alveolar formation. Within three months of the enucleation there was a recurrence within the orbit and Sir John Parsons carried out exenteration of the orbit. There was a mass of round-celled recurrent growth not definitely delimited from the orbital tissues. The stump of the optic nerve, in transverse section at the posterior limit, was free from growth cells. An extension of extra-bulbar sarcoma within the globe was an extremely rare condition. Less rarely, sarcoma started in the choroid, and extended out through the coats of the eye, usually by the perivascular lymphatics of the various perforating vessels. Mr. Neame thought there was more evidence that this growth was of extra-bulbar origin than that it was at first intra-bulbar. The reasons he adduced were:

(1) The earliest symptoms were referred to the epibulbar region above the cornea. (2) The vision in the eye, within two months of the commencement of symptoms, was $\frac{6}{9}$ with the appropriate glass; no visual failure in it was noted until 7 months after the onset. An extensive choroidal growth usually affected the vision. (3) The growth was much more massive at the point first noticed than elsewhere. (4) The gross appearance of the choroidal growth resembled a secondary or metastatic growth more than a primary growth there. (5) The alternative, flat sarcoma of choroid, was usually densely pigmented, whereas this was a non-pigmented growth. (6) Flat sarcoma was almost always relatively vascular, whereas throughout the present growth there were many delicate capillaries. (7) Flat sarcoma often invaded the deeper layers of the sclerotic, but this growth had only slightly done so, but had definitely invaded the superficial layers of this structure in various parts. He concluded with some extracts from literature bearing on the subject.

Sir John Parsons referred to the close mimicry of this condition, in its earlier stages, of gummatous infiltration of the conjunctiva and sclera, especially when, as in this case, the

reaction to Wassermann was positive. Only slowly, after a long course of anti-syphilitic treatment had been given, was it realized to be a new growth. Concerning epibulbar growths in general, on reading cases of the kind he could not quite convince himself that there had not been some small lesion in the eye which had subsequently extended outwards. Even in Mr. Neame's case there was that possibility, though the author of the paper had adduced much evidence favoring the opposite possibility.

Mr. MAYOU threw out the suggestion that a flat sarcoma might have existed in the anterior part of the globe and then perforated, good vision nevertheless persisting. In flat sarcoma the retina remained attached for a long time. One of his patients had detachment in the anterior part of the globe, and vision was still $\frac{6}{18}$ when the eye was removed. The growth had infiltrated the canals along the vortex of veins and lymphatics. The patient died two years later of recurrence. He suggested that this case should be submitted to a Pathological Committee, to place it on a sure basis. This was agreed to.

Mr. E. TREACHER COLLINS pointed out that there could be no doubt as to the direction of spread in the case of epithelioma as that type of growth never started in the eye, however much doubt might be thrown on the manner of spread of epibulbar sarcoma. He referred to a paper he read some years ago before the Ophthalmological Society on epibulbar epithelioma. He showed then illustrations of epithelioma spreading along the vessels at the limbus into the canal of Schlemm, and he had read of other cases which behaved similarly. A further point favoring Mr. Neame's view was the way the growth spread on the outer surface of the sclerotic. If a piece for diagnosis had been removed in this case earlier, removal of the eyeball might have been obviated, and the case rendered more hopeful. He referred to a case of his own in which he cut off the top of the growth and sent the patient to be further treated at the Radium Institute, and after two or three applications of radium, the growth entirely disappeared and he knew of no recurrence of it.

Mr. J. H. FISHER agreed that a piece should have been removed earlier, and referred to a case of his own of similar nature in which subsequent application of the actual cautery prevented any recurrence.

Mr. LESLIE PATON also alluded to an interesting case, in which the application of radium for 24 hours after the operation kept the man free for a long time, and a swelling of the soft palate, probably of the same nature, which developed was similarly treated with radium and that also disappeared.

The ordinary meeting of the Section was held on Friday, February 10, 1922, the President of the Section, Dr. JAMES TAYLOR, occupying the Chair.

Symmetrical Macular Disease.

Mr. FRANK JULER showed a youth with **symmetrical disease of the macula.** The changes were quite superficial in the retina, not in the choroid. The youth was sent up by his employers in a rubber factory, to find out whether anything could be done for him, and also to ascertain whether the condition had any connection with his occupation. He had been at the factory three years, his eye condition having appeared just before he started there. It was ascertained that the lad's work did not bring him into contact with the fumes given off in the manufacture, and Mr. Juler did not think there was any relationship of the condition to the employment. From the appearances it might possibly be a case of familial disease, such as Dr. Batten pointed out in association with cerebral degeneration; but this lad was quite bright and alert, and seemed to have done well at school. An elder brother of the patient was affected somewhat similarly, and he had been seen by Mr. Burdon-Cooper, of Bath, in 1915, when he was 21 years of age. Vision was $\frac{6}{6}$ in each eye, and there was central choroiditis. He was given some mercurial ointment for inunction, and he was seen again in 1919, when the vision was much worse, and Wassermann reaction had been positive on two tests. Some members of other branches of the present patient's family seemed to be affected by a similar condition. Another possibility was that this condition was inflammatory; but the boy appeared to be healthy, his Wassermann was negative, and it had remained so even under a provocative dose of Novarseno-bouillon. Von Pirquet was negative; the only feature out of the ordinary which was discernible was the presence of some septic crypts in the tonsils.

Plastic Operation for Lid Deformity.

Mr. OLIVER showed a patient on whom a plastic series was being done with the object of making a passable eyelid. The subject was a man who was wounded in 1916. When he came to the exhibitor two months ago he had had a number of operations. When he saw the man, the condition was that the whole of the skin of the upper lid had disappeared, and the lid was pulled up and was adherent to the upper orbital margin. The cornea was exposed, and there had been severe corneal ulcerations, which had left a nebula. He first excised all the scar tissue, and he thought that in all plastic operations it was most important to replace tissue in its normal situation. The tissues of the lid which were left could then be replaced, with some of the lashes. That left a large bare surface to be covered up, and it was covered by a temporal flap, in which flap it was very important to include the superficial temporal artery. All that now remained was to remove the whole pedicle. The operation was Major Gillies', and he had found it a most useful one.

Cholesterin Crystals in the Cornea.

Mr. R. AFFLECK GREEVES showed, as a curiosity, a patient with cholesterin crystals in the cornea; he had not seen or heard of such a case before. Cholesterin was common in other parts of the eye; in the retina and vitreous, and sometimes in the anterior chamber and lens. This patient had had extensive chronic ulceration of the cornea, with much photophobia and pain. There was found to be chronic disease of the antrum. After that had been treated, the ulceration cleared up a good deal and healed over. There were still attacks of pain and photophobia, but without ulceration. The patch of cholesterin had gradually increased in size for a year, and now there were also two smaller patches. The cholesterin probably resulted from the splitting up of the protein molecule, and, apparently, it could arise from the degeneration of blood, or from degeneration of tissues, though the usual form of degeneration was hyaline, with calcareous deposits.

Mr. M. S. MAYOU referred to one case he had seen of cholesterin in the cornea, and that had long-standing interstitial

keratitis. There were a number of vessels going up towards the patch, and he thought there had been some hemorrhage from these vessels into the corneal substance, which had given rise to the cholesterin. These deposits were more frequently associated with hemorrhage than with any kind of degeneration.

The PRESIDENT thought there must be some associated cause not yet determined, otherwise it must occur much more frequently.

Mr. GREEVES pointed out, in reply, that with the lessening of the blood vessels the cholesterin patch was getting bigger.

On Certain Structures Associated with the Upper End of the Choroidal Fissure.

Miss I. C. MANN read a paper, supplemented by models and an epidiascopic demonstration, on **the morphology of certain developmental structures associated with the upper end of the choroidal fissure.** The particular structures she was concerned with were (1) a small, cone-shaped mass of cells in the center of the optic disk in the human embryo of the 10th week and later; (2) the mass of unpigmented cells found just below the optic stalk on the outside of the eye in a six weeks' human embryo. She wanted to show how these structures were produced by a more rapid growth of the inner layer of the optic cup than of the pigment layer. She had arrived at the conclusion that, from this point of view, the eyes of vertebrates could be divided into those showing an overgrowth of the inner layer, and those which did not show it. In a six weeks' human embryo the pigment layer was thicker, and the inner layer was separated from it. She demonstrated a small mass of unpigmented cells which was continuous with the pigment layer, a point to which she attached importance. A section through the eye of a two-day chick showed a greater elaboration of structures connected with closure of the choroidal fissure. There was here a definite overgrowth of the inner layer, that layer being thrown up into folds; and in the region of the fissure the unpigmented inner layer was heaped up on the inner side, forming a ridge, and was everted in the lips of the fissure where the inner layer became continuous with the pigment layer. At this stage, in the bird's eye, the upper part of

the eye, above the insertion of the stalk, was bigger than the lower part, and the bird's eye grew by extension of the lower part below the insertion of the optic stalk. It was stated that the bird had not a definite hyaloid artery, but in very early stages one could demonstrate a mesoderm containing blood corpuscles, this strand passing across the eye, similar to the hyaloid artery in mammals. Miss Mann proceeded to show a large number of excellently prepared specimens from different animals and from the human embryo. It had been found that the hyaloid artery got on it a bulbous enlargement as it was passing through a mass of cells, and it was this budding out which vascularized the mass, and their insinuation among the nerve fiber layer formed definitive branches of the arteria centralis retinæ. The small mass of unpigmented cells on the outside of the embryonic eye was the remains of the portion of the inner layer which occasionally became everted in the upper part of the cleft, before it closed. If the eversion took place only at the upper end of the cleft, and no nerve fibers grew into it, as in man, the little mass after a time disappeared.

Dr. LEIGHTON DAVIES spoke of a case he had in which all the retinal vessels appeared at the periphery of the disk, just as in the rabbit's eye.

The PRESIDENT expressed the congratulations of the Section to Miss Mann for her interesting demonstration, and the beauty of the models and specimens with which she was working.

Three Cases of Choroidal Sarcoma.

Dr. HENRY J. MAY and Mr. F. A. WILLIAMSON-NOBLE contributed papers on this subject, Dr. May describing the clinical and operative details, and Mr. Williamson-Noble the pathological findings. All the three cases were seen by Dr. May within 19 days.

The first patient, a robust man of 60 years of age, complained of failing vision for near work, and discomfort with his glasses during a year. His distant vision was perfect. There was no evidence of inflammation in either eye. The tension in the right eye was a little fuller than in the left; the right pupil was slightly larger than the left, and was sluggish to light; the media were clear. In the fundus, below the outer side of the optic disk, he found a kidney-shaped swelling, meas-

uring 1½ disks one way, one disk the other; on its border were two small retinal hemorrhages. There was evidence of a pushing forward by a solid mass behind. The man had been getting thinner in the last 4½ months. At that time he would not consent to the enucleation operation which was urged. Seven months later he returned with an attack of acute glaucoma. The eye was then quite blind, and no view of the fundus could be obtained. Three days later the patient consented to enucleation. Mr. May found a typical mushroom-shaped growth, with a broad base, and it occupied the whole macula; the retina was detached. There was no evidence of extension of growth. On section the growth did not seem to have involved the sclerotic coat, and section of the optic nerve showed no sign of growth.

The second was in a man aged 52, very healthy looking. He had a painful red eye, but no trace of iritis. The cornea was bright and clear, and the tension plus 2. The anterior chamber was apparently obliterated, and the iris seemed to be in contact with Descemet's membrane. An opaque iridescent lens was tilted forward. On enucleating the eye he found a melanotic sarcoma the size of an average pea. As section of the optic nerve showed a suggestion of extension of growth to it, the orbit was exenterated. There were as yet no signs of recurrence.

The third patient was aged 67. He had been unable to see with the right eye for six months. In the right fundus, external to the disk, there seemed to be a large pigmented mass, with folds of retina lying over it or intermingled with it. The edge of the right optic disk was blurred and indistinct. Over four months later the man said his eye was totally blind; the pupil was dilated and inactive. In the fundus could be seen a large mass pushing the contents forward. Though 5 ft. 11 in. in height, this patient weighed only 8 st. 5 lbs. The eye was enucleated. The growth was a melanotic sarcoma, the optic nerve showed no extension of the growth to it. The patient had since had pains in the chest and abdomen, so there was reason to fear visceral involvement and a downward course. Dr. May discussed a number of general questions connected with this type of case, and Mr. Williamson-Noble followed with a demonstration of sections of the growth.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. CONRAD BERENS, Jr., Secretary.

MEETING OF DECEMBER 19, 1921. DR. L. W. CRIGLER, CHAIRMAN.

Presentation of Patients. Dr. W. H. Wootton presented a case of **paralytic miosis.** The patient, female, twenty-five years, came to the Manhattan Eye, Ear, and Throat Hospital complaining of sudden loss of vision in the right eye. Vision O. D. shadows, O. S. $\frac{20}{40}$ with correction. Marked iridodonesis and miosis O. U. There was a complete retinal detachment in the right eye. The lens in each eye could be made out in the lower part of the vitreous. Pupils could not be dilated beyond *2mm* with either atropin or cocain, no posterior synechiæ,—no ptosis or other signs of involvement of the cervical sympathetic, no glandular tumor in neck. Dr. Wootton thought that the miosis was probably due to a maldevelopment of the dilator fibres. This case had originally been one of myopia, the dislocation of the lenses, apparently spontaneous converting it into hypermetropia. There was no history of injury. Another rather interesting feature was that with her present correction, the best that could be given for distance, she could read Jaeger No. 3 at from 30 to 18*cm*. This apparent accommodation in an aphakic eye was not accompanied by any further contraction of the pupil. It should also be stated that the pupils reacted actively to light.

Dr. E. S. Thomson presented a case of **glaucoma following a cycloplegic.** The patient, L. H., 30, female, came to the clinic February 17, 1921, complaining of asthenopic symptoms. She was given a solution of atropin (two grains to the ounce)

and told to return for refraction. February 19th she returned with pupils well dilated, having put atropin in her eyes four times. Her visual tests were: R. V. = $\frac{10}{10}$ w + 2.25; L. V. = $\frac{10}{10}$ w + 2.75.

February 24th she returned stating that for several days past her eyes had felt bad. Examination showed both pupils still dilated, corneæ hazy, slight circumcorneal injection, and the tension elevated, being 60 (McLean) in each eye. She was treated with 1% pilocarpin and eserin (gr. ½ to 1 oz.) and her discomfort lessened, but the pupils did not fully contract or the tension remain normal. March 1st she was admitted to the hospital and kept more closely under observation, but without the desired effect. At times, in spite of all treatment, the tension would rise to 80. It would, at times, reach normal but would soon rise again. On account of the lack of severe symptoms, operation was delayed but a double iridectomy was finally done March 12th under a general anæsthetic. She recovered without incident and the tension has remained normal since. The vision is now $\frac{10}{10}$—in each eye with a correcting lens.

The question has often been raised in these cases as to whether any constitutional pre-disposition has existed which might have been a factor in causing the attack. The anterior chamber was not particularly shallow before the attack. She was referred to Dr. Houghton for a metabolic study.

Family history negative; recurrent attacks of tonsillitis and frequent nasal colds.

General physical examination: Well nourished young woman, of good color and general healthy appearance, with a possible slight sallowness of the skin. Blood pressure 155. Wassermann negative.

Urine negative except for indican and faint trace of albumin. Blood chemistry normal, except urea (40.32).

The tonsils and the nasal sinuses were found affected and were operated upon.

After the eye operation, there have been slight variations of tension without any significance. The attack of glaucoma was of unusually slow onset and while not of great severity, was certainly most intractable.

Discussion: Dr. Koller says that it is an old experience,

that a cycloplegic and even mild mydriatics as cocain or euphthalmin cause an outbreak of glaucoma in an eye predisposed to it. It is significant that a dilated pupil goes with prodromal, glaucomatous attack, and that states of hypotension are generally accompanied by a narrow pupil. This connection is by no means satisfactorily explained by the Knies-Weber theory of glaucoma, and the question is, why a dilated pupil necessarily goes with a glaucomatous attack. Dr. Koller refers to the paper which he read at the last meeting of the American Ophthalmological Society on "The Physiological Mode of Action of Mydriatics and Miotics—Explaining their Effects in Hypertension," in which he showed that all mydriatics have a powerful constricting effect and all miotics have a powerful dilating effect on the iris vessels, congesting the same; an effect to which in part dilatation of the pupil (by shrinking of the iris) and vice versa contraction of the pupil (by unfolding and stretching of the iris) is due. In the prodromal or acute glaucomatous attack we have before us a state of œdema of the anterior part of the eye, accompanied by a stagnant circulation in the ciliary system. Proof of this stagnation is relative anæmia of the iris (dilated pupil) and engorged ciliary plexus (dilated anterior ciliary veins—their collaterals). This stagnation of the circulation is relieved by the miotics (including dionin), and also by all other agencies making for a congestion to the head, for instance, sleep, administration of morphin, caffein, etc.

Speeding up the circulation causes lessening of the œdema, thus relieving the hypertension. The stagnation of the circulation is aggravated by all mydriatics, because they contract the iris arteries, and this explains their adverse influence in glaucoma already existing, and perhaps also their precipitating attack in an eye disposed to glaucoma.

But the beneficial effect of the miotics, and the adverse effect of the mydriatics is not confined to the "inflammatory" variety of glaucoma, which is a sort of angioneurotic œdema, caused by an unknown agency. All kinds of hypertension, whether primary (chronic simple glaucoma) and thus of vascular origin, or secondary caused by lens cortex or vitreous body, clogging the circulation after an operation or traumatism, is equally affected by mydriatics and miotics, thus furnishing

additional evidence of the correctness of the theory here propounded.

Doctor HARTSHORNE said he had seen two cases of glaucoma following a cycloplegic: 1. S., 40 years old. Used homatropin cycloplegic twice without ill effect. In 1921 four days after the use of the cycloplegic homatropin, he returned for post cycloplegic examination with acute glaucoma O.D. and O.S. normal. Before cycloplegic had been used fundi and tension were normal (with fingers). Did not respond to medical treatment and after iridectomy had intraocular hemorrhage,—final and present vision $\frac{2}{40}$ with marked cupping of disc. He added in discussion that two drops of pilocarpin 1% had been put in the eyes to counteract the homatropin. Eight years previously he had done an iridectomy for glaucoma on the father.

2. The record of a woman of thirty in whom he did double iridectomy for chronic glaucoma, showed that he had 1 year previously used homatropin without ill effect.

Dr. ROSE said, as the administration of a cycloplegic causes the iris to press towards the limbus of the eye, interfering with the free circulation of the blood, lymph and aqueous, in their respective channels, is it not possible that we may be adjudged liable, in a case like this one of Doctor E. S. Thomson's? Would it not be well to make it a daily practice to administer at least two doses of ½% eserin before allowing a patient to leave the hospital or office, after administering a cycloplegic?

Doctor C. H. MAY said he rarely uses atropin in the estimation of errors of refraction; children, particularly squint cases, offer occasional exceptions. His routine plan is to employ a 2% solution of homatropin with a 1% solution of cocain; of this mixture, one drop is instilled in each eye every few minutes for four doses; the patient is instructed to keep the eyes closed for one hour and is then ready for the examination. The effects of the homatropin are neutralized with eserin in every case; one drop of ½ of 1% solution is instilled and this is followed by a similar instillation after a few minutes; the patient is informed of the object of this and of the fact that there follows a somewhat uncomfortable twitching of the lids from the effects of the eserin; unless this is explained there is apt to be nervousness and excitement on the part of the

patient after he leaves the office. The result of these instillations is that the patient is able to resume his usual occupation after a half hour or an hour, whereas, if the neutralizing drops are omitted, the dilated pupils and interference with accommodation will last two days. Dr. May had never encountered a case of glaucoma as a result of a mydriatic or a cycloplegic and he thought that the probable absence of such an accident was due to the regular use of eserin as described.

Dr. COHEN suggested the advisability of making tonometric rather than digital examination of the eye-ball in cases where there existed the slightest possibility of an increase in the intraocular pressure.

Dr. CLAIBORNE said his experience in this matter was practically parallel with that of Doctor May. During that time it had been his custom to use atropin between childhood and thirty-five years—afterward homatropin between thirty-five and forty-five. He had never seen a case of glaucoma following either and he had never used pilocarpin or eserin to cut short the duration of the mydriasis.

Dr. T. H. CURTIN reported three cases of **tumor of the eye-ball and of the orbit.**

Case 1. Melano-sarcoma of choroid and ciliary body in a woman aged 48. She had noticed failing vision in the right eye for the past three months. Vision R. E. Fingers to nasal side; L. E. $\frac{20}{100}$ −2.00 cyl, ax 60 = $\frac{20}{20}$.

The right eye showed what appeared at first to be an iridodialysis at about 3 o'clock, but on more careful examination proved to be degeneration of the iris, with black pigment showing through. The ophthalmoscope revealed a black mass back of the lens, encroaching half way to center of pupil. Tension—McLean tonometer R. E. 45; L. E. 30.

The eye was enucleated.

The specimen presented was the nasal half of the right eye, revealed a black tumor mass occupying in its entirety the anterior one half of the vitreous chamber, involving the ciliary body and iris and pressing against posterior surface of lens.

Case 2. Sarcoma of orbit. Operation—Exenteration. P. W., age 79. Came to hospital on February 3, 1921, for pain in left eye and a sticking together of the lids, also states that he had a slight bulging of the left eye, but could see fairly well

with it, the proptosis and loss of vision gradually increased, and September 1, 1921, he had proptosis of the left eye with limitation of movement of the globe in all directions. No light perception, and a tumor mass could be felt, particularly on outer aspect of orbital cavity.

X-ray Report—Negative. At operation, complete exenteration of the left orbital cavity was done. The eye with entire tumor mass and periosteum lining the orbital cavity was removed en masse, the tumor was found to have perforated into the ethmoidal cells and into the middle meatus of the nose and maxillary sinus. The skin of the lids after scalping of the cilia was inverted into the base of the orbital cavity and used as skin flaps, these have grown so well that the cavity at the present time is nearly completely lined with skin, except for the two openings, viz.—into ethmoidal cells and into nares, and also into maxillary sinus where the openings are much larger than at the time of operation.

This patient received two radium exposures, each of 20 hours' duration, at Radium Institute, the first, two weeks after the operation and the second, the following two weeks. At the present writing there is no evidence of any return of the growth, the left orbital cavity is nearly completely lined with skin, and it is contemplated to close opening into nares by a pedicle skin graft, sliding it downward.

The specimen as presented shows a tumor surrounding the optic nerve, and the posterior segment of the eye-ball, this before operation filled the entire orbital cavity. There appears to be no extension of the tumor into the globe and although surrounding the nerve intimately, the tumor probably originated in the retro-orbital tissues. Microscopically—mixed cell sarcoma.

Case 3. Leucosarcoma of choroid. W., age 29 years. On Jan. 5, 1918, vision was $\frac{20}{20}$ in each eye and fundi were normal. On Dec. 13, 1920, he complained of a blurred vision in right eye. Vision O.D. $\frac{20}{30}$, O.S. $\frac{20}{15}$.

In the right eye a small white spherical detachment was seen above and out in the periphery of the right fundus.

The intraocular tension was 30 in each eye. Wassermann—negative. Gonorrheal fixation—negative. Koch subcutaneous tuberculin test—negative.

I advised early enucleation but the patient insisted on taking radium treatment. One exposure was given with no apparent effect except slight reddening of surface of tissues. April 12, 1921, at Bronx Eye and Ear Infirmary enucleated R. E., normal recovery.

Specimen shows a white tumor, 15 × 12*mm* occupying the entire anterior segment of the vitreous chamber, in direct contact with the posterior surface of the lens—with no involvement of the lens or iris.

Pathological diagnosis—Spindle-cell sarcoma.

DISCUSSION: Doctor GOLDSTEIN stated that they had two cases of Orbital Tumor on Doctor Wheeler's service at the New York Eye and Ear Infirmary for which an eviceration of the orbit was done at the same time a Thiersch skin graft was applied on rubber tissue to the bare bone. This was then held in place by a Mikulicz tampon. This dressing was left *in situ* for one week. In one of the cases there was a complete take. In the other ⅔ of the graft had taken.

This method which is Van Noorden's, has certain advantages. It shortens the stay at the hospital, leaves a perfectly smooth surface, and if a recurrence is taking place it can be readily seen, whereas if granulation tissue is permitted to grow it obscures the new growth.

Doctor R. C. DODD reported a case of **keratitis punctata superficialis.** J. W., age 25.

In July of 1919, patient had redness, pain, and watering of the left eye. This persisted for months before he sought treatment and then only because the same condition began to develop in the other eye.

He was treated at various clinics and came under my observation in Nov., 1921, complained of intense photophobia, watering, and pain in both eyes. On examination he showed numerous superficial punctate opacities on each cornea—with one superficial ulcer on his left cornea. No vascularity of the cornea and no deeply situated opacities. On examination of the cornea under the loupe the areas were seen to be sharply defined rounded spots with a central "nucleus" over which the cornea was unbroken and depressed rather than raised.

Wassermann negative, urine negative, nose and throat dep't report enlarged turbinates—deviated septum and a small

amount of pus in the tonsillar crypts. Skin lesions due to acne and at present under treatment at the Vanderbilt Clinic. Tuberculin intradermal test showed a moderately positive reaction (2+) in dilutions of 1–10,000 and 1–1000. Not sufficiently positive to justify tuberculin treatment.

The treatment has consisted of atropin, heat, yellow oxide ointment and quinine grs. 5 t.i.d. with protective smoked lenses.

The case presented shows some variations from the classic description, possibly due to the length of time that the process has been active—(18 months). These variations are: (1) Ulcer formation in left eye, which is now healed. (2) The absence of elevation of epithelium over the areas of infiltration —in fact the epithelium over many areas seems actually recessed. (3) The large size of some of the areas with their peculiar appearance of a central nucleus.

DISCUSSION: Doctor W. B. Weidler does not believe this is a case of keratitis ex acne rosacea, as was suggested, for there is no marked inflammation of the cornea and the periphery of the cornea is not involved.

He has seen a number of cases of keratitis ex acne rosacea and there is often perforation of the cornea with prolapse of iris into the ulceration. A tendency to chalazion formation has been reported by von Hippel.

Dr. DODD reported a case of **retinitis.** P. C. age, 16, has never seen well with her right eye. In April of this year the vision in the right eye was fingers at 2 feet (myopia 19D). The vision in the left eye was $\frac{3}{60}$ made $\frac{3}{20}$ by -1.00×10. which glass was ordered. Fundus normal.

In October she complained of blurred vision in her good eye which had come on rather suddenly a few days before. Her vision was $\frac{20}{100}$ and the fundus showed an extensive area of retinitis extending from the disc to the macula, the retina was œdematous and raised about 2–3 diopters. In the extreme periphery there were some other areas of retinitis.

Physical examination negative. Wassermann negative. Urine negative. X-ray of sinuses negative.

Intra-dermal tuberculin test showed a strongly positive 4+ reaction in dilution of 1–1,000. Tuberculin injections begun November 16 with 3 minims of B.E. 1–10,000. In the course

of 2 to 3 weeks the retinitis subsided leaving distinct clear-cut whitish bands resembling retinitis striata, situated below the vessels and extending from the region of the disc towards the macula. At this time an area of proliferating retinitis was seen extending from the macular region forward into the vitreous and some fresh hemorrhages in the periphery of the fundus below where an extensive retinitis still persisted.

On December 20, 1921, the striate bands appear to have disintegrated into rounded spotty areas totally different from the clear-cut white bands that were seen previously. The proliferating strands extending into the vitreous are well seen —as is also the extensive retinitis with hemorrhage in the periphery below and to the temporal side.

There are certain factors in this case that strongly resemble the disease known as Coats' disease of the retina or massive exudative retinitis.

Doctor UNGER showed two cases of **keratoconus.** CASE I. C. S., age 22 years, always nearsighted, influenza 1918, since then eyes rapidly worse. Vision O.D. $\frac{4}{200}$, O.S. $\frac{3}{200}$, when first seen was wearing O.D.–6.50 cylinder, axis 165°; O.S.–6.50 cylinder, axis 180° which gave vision of $\frac{20}{100}$ right and $\frac{20}{200}$ left. Under homatropin, October, 1920, O.D.–0.50 spherical ⊃ –5.00 cylinder, axis 45° gave $\frac{20}{40}$ plus 3; O. S.–4.50 spherical ⊃ –8.00 cylinder, axis, 5° gave $\frac{20}{70}$. One year later, October, 1921, homatropin O.D. –4.50 spherical ⊃ +8.00 cylinder, axis 135°, gave $\frac{20}{40}$ +3, O.S. –2.50 spherical ⊃ —11.00 cylinder, axis 5°, gave $\frac{20}{70}$.

CASE II. D. L., always nearsighted. Vision O.U. $\frac{5}{200}$ under homatropin February, 1921, O.D. +3.00 spherical ⊃ —12.00 cylinder, axis 75°, and O.S +4.00 spherical ⊃ —8.00 cylinder, axis 85°, gave each eye $\frac{20}{80}$ and both $\frac{20}{80}$ +3.

Doctor WEIDLER reported a case of **altitudinal hemianopsia due to occlusion of the inferior temporal artery.** F. C., age 48, on Oct. 20, 1921, drank some "liquor" and a few days later he noticed difficulty in seeing above with the left eye. Six weeks later he came to the Manhattan Eye and Ear Hospital.

Pupils 3*mm*, react, tension normal, vision O.D. $\frac{20}{40}$, O.S. $\frac{20}{80}$. The ophthalmoscopic study of the fundus at this time showed no changes except a slight pallor of the lower half of the disc. Fields showed a complete altitudinal hemianopsia

except for a small portion of the upper outer field. About ten days later, ophthalmoscopic study of the fundus revealed the lower half of the disc decidedly paler and whiter showing well advanced atrophy. The vessels were all normal in size, shape and color, except the inferior temporal retinal artery, which was contracted. On the disc and at some distance from the disc there is a distinct narrow white line along the vessel wall, showing a partial or complete obstruction of the vessel with secondary obliteration of the vessels. The process is gradually extending along the length of the vessels. The field remains about the same, with increasing atrophy of the disc.

Dr. WEIDLER reported a case of **paralysis of the third, fourth, and sixth nerves** involving all of the extraocular muscles with partial ptosis of the left eyelid. S. R., age 16. Vision O.S. $\frac{8}{200}$ (central corneal opacity).

In November, 1921, struck over the left eye and brow with a hand, which made her "see stars" but later could see better. The next morning when she awoke diplopia noted. After breakfast she was nauseated and vomited, and the same thing occurred the following two mornings. She was unable to resume work, and came to the Manhattan Eye and Ear Hospital, December 3, 1921.

Examinations: O.D. corneal opacity central with nystagmoid movements of the eye, which increased greatly as time went on; vision $\frac{8}{200}$ O.D. $\frac{20}{20}$. There was slight ptosis, which never progressed and paralysis of all of the muscles except the external rectus. Field normal. Urine analysis and Wassermann tests were negative. The X-rays of sinuses showed deviation of the septum to the left with some involvement of the posterior sinuses. Three days later the external rectus became paralyzed. There was a feeling of numbness of the skin over the left forehead and head with complete loss of sensation for heat and cold.

Ten days later movement in the external rectus restored to two-thirds of normal, with slight power of elevation and depression.

The paralysis of the ocular muscles was probably due to a blow which fractured the ethmoid plate of the orbit and was followed by a hemorrhage in the orbit, or there may have been a hemorrhage into the middle fossa of the skull.

The treatment prescribed was hydrarg., bichlor. gr. $\frac{1}{10}$ and potassium iodid gr. X, massage over forehead and scalp. At the last examination there was almost complete action of the external rectus, increased power of elevation and depression, and it would seem that there would be nearly a complete return of motion in all the muscles.

Dr. Ben Witt Key showed a case of **fat implantation.** C. B., man, 24 years of age, when 8 years old was struck by the end of a wire, cutting and penetrating the cornea of right eye. On April 30, 1921, O.D. no L.P., slightly smaller than normal, divergent, and a menace to the other eye.

Operation was performed on May 8, the four recti muscles being attached in the conjunctival sulci according to the method which I suggested and reported in detail before this Society, December 19, 1921.

There is now sufficient prominence of the prothesis to conform to that of the other eye. The excursions of rotation are all that could be expected of a prothesis, but more important still is the sensitiveness to motion concomitant with the slightest motion of the other eye. The orbit is filled with the fat implant together with well-formed conjunctival sulci, temporalward as well as nasalward, without absorption over a period of 7½ months.

This result is due to:

1. The recti muscles are sutured at the sides of the fatty mass and in the floor of the conjunctival cul-de-sacs and, therefore, traction is not made upon the implant but upon the conjunctival attachments of the lids and canthi, thereby allowing normal growth of the fat in Tenon's capsule.

2. Tenon's capsule is entirely filled with one piece of fat from the front of the thigh.

3. The bleeding orbit is not to be irrigated with any solution, nor should bichloride or other cotton pledgets be forced into it. On the contrary pieces of gauze saturated in warm salt solution are placed firmly in the bleeding orbit while the fat is being taken from the thigh. Unusual bleeding is checked before inserting the implant, but the implant is not taken from the leg until the orbit is properly prepared for it.

4. The bandage is firmly applied, the other eye bandaged (when the patient has become entirely conscious from an-

æsthesia) and should remain bandaged for four or five days—; dressing every two to three days with gauze saturated in warm salt solution, rubber tissue being used to retain the moisture.

DISCUSSION: Doctor WEIDLER did a series at the Manhattan Hospital and a third to a half of fat was absorbed. It may have been due to the operation he did. He never lost an implant by sloughing, or infection. He will be glad to try Doctor Key's operation in the future.

III.—**The Anatomy of the Human Orbit and Accessory Organs of Vision.** By S. Ernest Whitnall, Professor of Anatomy, McGill University, Montreal. 8vo., pp. 428. 195 illustrations. Oxford University Press, 35 West 32d Street, New York. Price $10.

This book arose from lectures given to candidates for the Oxford Diploma of Ophthalmology, and the many dissections and preparations which were used in those lectures are now reproduced by photographs, thereby making this important subject available for general study.

The subject is divided into: a. The bones forming the orbit and their important relations to the nasal accessory cavities; b. The eyelids, conjunctiva, and lacrimal apparatus; c. The contents of the orbit: the globe, ocular muscles, blood vessels, and nerves; d. Appendix: the cerebral connections of the nerves, bibliography, and index.

The description of the cerebral course of the nerves related to the eye, while not necessarily belonging to the orbit, completes the anatomical subject and adds greatly to the usefulness of the book. A complete bibliography of the anatomical articles and books published since 1900 will be of value to investigators.

In the careful and complete elaboration of a difficult subject, Professor Whitnall has placed ophthalmologists under a great obligation; his book should be in the library of every specialist.

A. K.

IV.—**Cataract and its Treatment.** By Lieut.-Col. H. Kirkpatrick (Ret'd), London, late of Madras, India. Pp. 201.

Illustrated, London. Oxford University Press, 35 West 32d Street, New York. Price $3.75.

Lieut.-Col. Kirkpatrick has given us a short but complete book on cataract, beginning with the anatomy of the lens, its changes in cataract, ætiology of cataract, varieties, symptoms, diagnosis, and finally its treatment by operation. Objections to the intracapsular operation are given as "tissue disturbance," a large percentage of vitreous loss, and adhesions of the iris and vitreous to the wound, which may cause late infections, late attacks of irritation, and glaucoma. For the young surgeon particularly extraction with capsulotomy is safest, and irrigation is regarded as an invaluable aid. The preparation of the patient, the operation, the after-treatment and complications, are carefully described, and give evidence of the writer's large experience and good judgment. The book can be recommended as a safe and conservative guide.

A. K.

V.—**Diseases of the Eye.** By Dr. Geo. E. De Schweinitz. Prof. of Ophthalmology, Univ. Pa. IX. edition, 832 pp. 415 illustrations and 7 colored plates. Phila. and London. W. B. Saunders Co. 1921. Price $10 net.

The new edition of this well known text-book has been carefully revised and new subject matter has been added, dealing with new observations of clinical, therapeutic, and operative nature. Important chapters have been rearranged and enlarged. The book is thoroughly up to date and remains one of the best text-books on the eye in any language.

A. K.

VI.—**Protein Therapy and Non-Specific Resistance**. By Dr. William F. Petersen, Associate in Pathology, Univ. of Illinois, Chicago, with an introduction by Dr. Joseph L. Miller, Univ. of Chicago, Pp. 314. 1922. The Macmillan Company, New York. Price $4.50.

This book is a comprehensive yet conservative review of the literature relating to non-specific therapy. It begins with a brief history of the development of this new mode of treatment, followed by a description of the various chemical

substances—mostly proteins—which have been employed to bring about non-specific reactions.

At the very outset, and repeatedly throughout the book, Dr. Petersen emphasizes the fact that the theory of non-specific immunization does not in any way contradict the theory of specific immunization; that non-specific therapy does not take the place of specific therapy, but that there are definite indications for each. Each mode of treatment is based on the reaction of the body to injury, and this reaction, Dr. Petersen states, is fundamentally similar under all circumstances, no matter how produced, and obeys all the commonly observed laws of biologic reactions.

His analysis and description of the non-specific reaction is clear and concise. A separate chapter is devoted to a description of the focal reaction—with especial emphasis on the focal reaction in tuberculosis.

The several theories attempting to explain the mechanism of the reaction are given, and the probable nature of the mechanism is explained. The mechanism of recovery in pneumonia, tuberculosis, and typhoid is gone into at some length.

An interesting chapter is devoted to the various skin reactions to infective and protein sensitization, and the probable rôle played by the skin in immunization.

In ophthalmology non-specific protein therapy has been of greatest value in phlyctenular conjunctivitis, trachoma, interstitial keratitis, suppurative keratitis, and iritis. Milk, typhoid vaccine, and diphtheria antitoxin were the proteins most commonly used in the treatment of these diseases.

The book is thoroughly scientific and up-to-date, placing non-specific therapy on a rational basis, and clearly demonstrating, in our opinion, the value of this new mode of treatment.

A. H. T.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

PRESIDENT: J. HERBERT FISHER, M.B., F.R.C.S.

ANNUAL CONGRESS, 1922.

The next Annual Congress of the Society will be held on Thursday, Friday, and Saturday, 11th, 12th, and 13th of May, 1922, at the Royal Society of Medicine, 1 Wimpole Street, W. I.

The arrangements will be as follows:

THURSDAY, MAY 11th.

MORNING

10 A.M. to 12.30 P.M. ...	... Presentation of Edward Nettleship Prize. Papers.

AFTERNOON.

2.15 P.M.	Discussion on "Industrial Diseases of the Eye (Excluding Accidents, Miners' Nystagmus and Glass-blowers' Cataract"). To be opened by Dr. T. M. LEGGE, C.B.E., His Majesty's Medical Inspector of Factories, and Mr. BERNARD CRIDLAND.
4.30 P.M.	Tea.
5 P.M.	Business Meeting. Members will dine together in the evening.

FRIDAY, MAY 12th.

MORNING.

10 A.M.... ... ... Papers.

AFTERNOON

2.30 P.M. ... ... **Visit to St. Bartholomew's Hospital, West Smithfield, E.C.1.**

Clinical Meeting. Members are invited to show cases of interest.

EVENING.

8.30 P.M. ... ... Tuberculosis of the Eye—Pathological Demonstrations with epidiascope and microscopes. Members are invited to show illustrations, specimens, and sections.

SATURDAY, MAY 13th.

MORNING.

10 A.M. ... ... Papers.

Members desirous of reading Papers, showing Cases or Specimens, or taking part in the Discussions, are requested to communicate as soon as possible with the Hon. Secretary, 14, Portland Place, London, W. 1.

Papers and Communications, subject to the judgment of the Council, will be printed in full in the *Transactions*.

Under the By-laws readers of Papers must not exceed twenty minutes, subsequent speakers ten minutes. The openers of the Discussions are allowed twenty minutes.

All Communications must be type-written.

J. F. CUNNINGHAM
F. A. JULIER } **Hon. Secs.**

OXFORD OPHTHALMOLOGICAL CONGRESS.

Master, SYDNEY STEPHENSON.
Deputy Master, PHILIP H. ADAMS.
Hon. Treasurer, SIR ANDERSON CRITCHETT, BART., K.C.V.O.
Hon. Secretary, BERNARD CRIDLAND, Salisbury House, Wolverhampton.
Past Master, THE LATE ROBERT W. DOYNE.

February, 1922.

PRELIMINARY NOTICE.

The Oxford Ophthalmological Congress will assemble at Keble College, Oxford, on the evening of **Wednesday, July 5th, next,** and the Meeting will be held on **Thursday, July 6th,** and **Friday, July 7th,** with an extension to the morning of **Saturday, July 8th,** should the programme of the proceedings demand it.

On Thursday, July 6th, a discussion on "THE SIGNIFICANCE OF RETINAL HÆMORRHAGES" will take place, to be opened by Dr. C. A. Hawthorne (London) and Mr. P. H. Adams (Oxford).

Members intending to take part in the Discussion are requested to kindly send in their names to the Hon. Secretary at their early convenience.

The Doyne Memorial Lecture will be delivered on the morning of Friday, July 7th, by J. Burdon Cooper, Esquire, M.D., F.R.C.S.E., the subject being "THE ETIOLOGY OF CATARACT."

The Official Dinner of the Congress will take place on the evening of Thursday 6th, in the Hall of Keble College.

A General Meeting will be held during the Congress at a time of which due notice will be given in the final programme.

It is hoped that members will contribute to the success of the Meeting with papers, pathological specimens, new operations, cases, or novelties of any kind.

Notification of such at the earliest opportunity to the undersigned will be appreciated.

BERNARD CRIDLAND,
Hon. Secretary.

Salisbury House,
Wolverhampton.